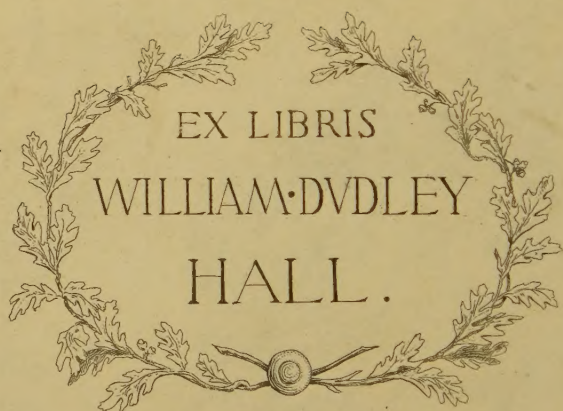




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OPHTHALMIC SCIENCE AND PRACTICE



A HANDBOOK OF
OPHTHALMIC SCIENCE
AND PRACTICE

BY

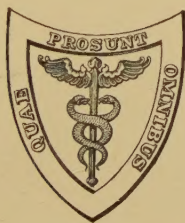
HENRY E. JULER, F.R.C.S.

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ROYAL WESTMINSTER OPHTHALMIC HOSPITAL; CONSULTING OPHTHALMIC
SURGEON TO THE LONDON LOCK HOSPITALS

WITH ILLUSTRATIONS

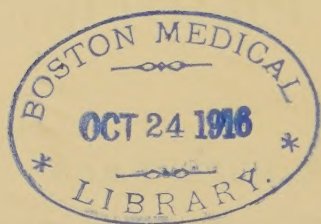
Third Edition

REVISED AND ENLARGED



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PREFACE
TO
THE THIRD EDITION.

IN the Third Edition of this work I have to thank my friend
Mr. G. S. KEELING for considerable help in the revision of
the text and in the drawings of microscopical specimens.

HENRY JULER.

23 CAVENDISH SQUARE, W.

PREFACE

TO

THE SECOND EDITION.

IN the Second Edition of this work considerable alterations and additions have been made both in the text and in the illustrations.

I regret that Mr. ADAMS FROST has not been able to assist me with the chapter on Refraction, but his place has been taken by Mr. JOHN GRIFFITH. I have to thank Mr. GRIFFITH also for much help in the preparation of the Photomicrographs, Original Drawings, and the derivations of Ophthalmic Terms to be found in the Index ; and Mr. TILLINGHURST ATWOOL for many new Coloured Drawings of the Fundus Oculi.

HENRY JULER.

23 CAVENDISH SQUARE, W.

PREFACE

TO

THE FIRST EDITION.

IN the preparation of the following work, it has been my endeavour to produce concise descriptions and typical illustrations of all the important affections of the eye.

With one exception, the coloured plates have all been taken from cases met with in the course of clinical work, chiefly at the Royal Westminster Ophthalmic Hospital, St. Mary's Hospital, and the Royal London Ophthalmic Hospital, Moorfields. With regard to the drawings of these and the other illustrations, I have received valuable suggestions and assistance from Mr. E. NOBLE SMITH.

The chapter on Refraction has been jointly written by my colleague Mr. ADAMS FROST and myself, and that on Colour-Vision is entirely his work.

My best thanks are due to my friend and colleague Mr. ANDERSON CRITCHETT for the kind way in which he has allowed me to make use of any cases coming under his, or our joint, care at St. Mary's Hospital, and for many valuable practical suggestions as to diagnosis and treatment.

I also have to thank Dr. E. J. EDWARDES for considerable help in the chapter on the Optic Nerve and Retina, more especially with regard to the views of Continental writers.

Finally, I am indebted to Mr. ADAMS FROST and Mr. ARTHUR K. WILLIS for their valuable help and suggestions in passing the book through the press.

77 WIMPOLE STREET, CAVENDISH SQUARE, W.

May 1884.

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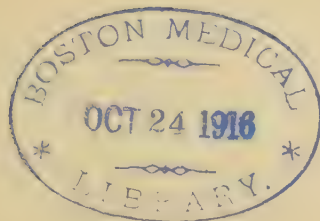
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A HANDBOOK OF OPHTHALMIC SCIENCE AND PRACTICE.

CHAPTER I. *THE EYELIDS.*

ANATOMY AND PHYSIOLOGY—BLEPHARITIS—PHTHEIRIASIS—CEDEMA—HORDIOLUM—CHALAZION—HERPES OPHTHALMICUS—CONCRETIONS—TRANSLUCENT CYST—DERMOID, CYST—CONGENITAL CYST—NÆVUS—XANTHELASMA—RODENT ULCER—PAPILLOMA—MOLLUSCUM CONTAGIOSUM—SARCOMA—LYMPHANGIOMA—LIPOMA—FIBRO-ADENOMA—MOLLUSCUM FIBROSUM—NEUROMA—CHANCRE—GUMMA—BLEPHAROSPASM—PARALYSIS OF ORBICULARIS PALPEBRARUM—PTOSIS—TRICHIASIS—DISTICHIASIS—ENTROPION—ECTROPION—EPICANTHUS—COLOBOMA PALPEBRÆ.

THE eyelids are muco-cutaneous folds especially constructed for the protection of the eyeball. They not only serve to prevent the entry of foreign bodies into the palpebral sac, but by their rapid action (reflex nictitation) remove particles of dust, mucus, &c., from the cornea and ocular conjunctiva. They assist also in keeping the cornea moist, and therefore transparent. If the act of blinking be suspended for a time, the exposed corneal epithelium will become dry and lustreless and the visual acuity reduced. This movement also aids the passage of tears into the lachrymal sac. At each closure the sac is pulled upwards and its cavity expanded, which causes the tears in the lacus lachrymalis to be drawn by suction into it through the canaliculi. When closed the eyelids, lubricated along their surface of contact by Meibomian secretion, convert the palpebral sac into a water-tight compartment.

ANATOMY AND PHYSIOLOGY.

From without inwards each eyelid presents the following structures (fig. 1) : skin, connective tissue, sphincter orbicularis and the ciliary muscle of Riolanus, the glands of Moll, a loose cellular interval with the expansion of the tendon of the levator palpebræ muscle, the tarsus with the Meibomian glands and gland of Waldeyer, the muscle of Müller, and the conjunctiva. The *skin* is continuous with that of the face ; at the free border of the lid it becomes con-

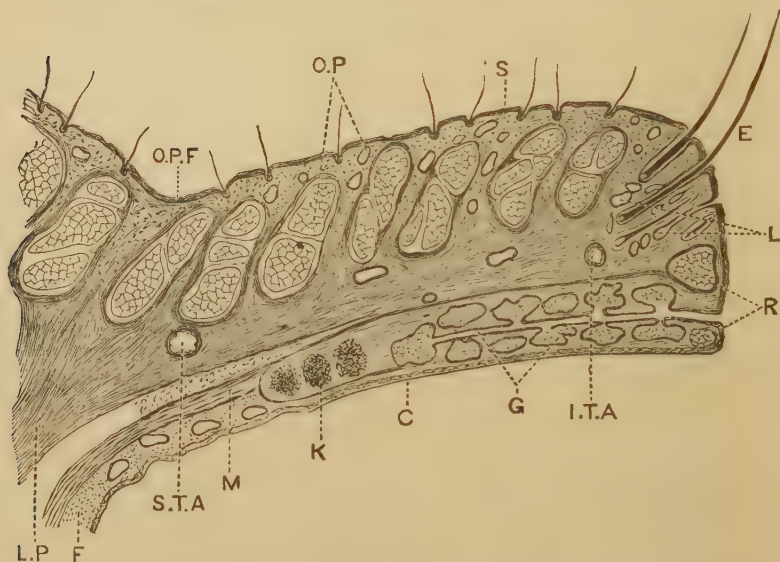


FIG. 1.—Section through Upper Eyelid (semi-diagrammatic).

O.P.F., orbito-palpebral fold ; O.P., orbicularis palpebrarum ; S, skin ; E, eyelashes ; L, glands of Moll ; R, muscle of Riolanus ; I.T.A., inferior tarsal arch ; G, Meibomian gland ; C, conjunctiva ; K, gland of Krause ; M, muscle of Müller ; S.T.A., superior tarsal arch ; F, fornix ; L.P., tendon of levator palpebræ.

tinuous with the conjunctiva. It is delicate, but otherwise resembles the general integument ; it is freely movable on the subjacent connective tissue. The *eyelashes*, or *cilia*, are slightly curved, and are placed in from two to four rows in the skin at the anterior border of the lids ; into the follicles open sebaceous glands—the *glands of Zeiss*. Between and behind the cilia are found the *glands of Moll*, modified sweat glands, situated deeper than the follicles of the eyelashes, pyriform in shape, and consisting of a dilated fundus twisted upon its long axis, from which a narrow duct passes to

the surface; the gland is lined with columnar epithelium, the duct with cuboidal cells, and its expanded mouth with stratified epithelium. The subcutaneous connective tissue is devoid of fat-cells, and communicates with the subconjunctival tissue by a plexiform mesh-work, in which the fibres of the sphincter orbicularis are embedded. The palpebral portion of the *orbicularis palpebrarum muscle* consists of thin, pale, and slightly curved fibres, surrounding the fissure between the eyelids. It is closely adherent to the skin by fine connective tissue, but glides loosely over the tarsi. It takes its origin internally by two heads, tendinous and muscular, from the lips of the lachrymal groove. The anterior head of origin is the *tendo oculi* (internal palpebral ligament), which is attached to the orbital ridge on the nasal process of the superior maxilla (*crista lachrymalis anterior*); and the posterior, usually considered a separate muscle, is the *tensor tarsi*, or *Horner's muscle*, which arises from the ridge on the lachrymal bone (*crista lachrymalis posterior*). The muscular head, or *tensor tarsi*, divides externally into two bundles, which are mainly inserted into the inner extremities of the upper and lower tarsal plaques. Some of its fibres, however, blend with the orbicular bundles which arch outwards from the *tendo oculi*. Surrounding the palpebral fissure the deeper fibres of the *orbicularis palpebrarum* meet in a fibrous *raphé* (external palpebral ligament); the superficial fibres curve round and intermingle with each other. Some muscular fibres surround the lachrymal sac, and others the canaliculi. The natural tone of the muscle suffices to keep the eyelids comfortably approximated to the eyeball, and the *puncta lachrymalia* inverted into the *lacus lachrymalis*.

The *tendo oculi* divides into two processes, an upper and lower, forming a Y-shaped ligament, one to each tarsus. At its point of division it is retracted slightly by the attachment of the palpebral aponeurosis.

The *tarsi* (tarsal cartilages) are two thin elongated plates composed of dense fibrous tissue. They give firmness and shape to each eyelid. They are situated beneath the fibres of the sphincter orbicularis muscle. The upper tarsus is somewhat oval in shape, and is thickest at its free border; its anterior surface receives some of the tendinous fibres of the *levator palpebræ*, and into its superior border the muscle of Müller is inserted. The lower tarsus is thinner, narrower, and of nearly uniform breadth throughout. The tarsi are fixed by fibrous tissue internally to the nasal process of the superior maxilla by the *tendo oculi*, externally to the malar bone, and above and below to the margin of the orbit by the *palpebral aponeurosis*. This fascia, which, with the tarsi, constitutes the *septum orbitale*, is,

in the upper eyelid, attached to the margin of the orbit above, below to the levator tendon and tarsus, at the outer side to the external palpebral ligament, but internally is attached to the crista lachrymalis posterior, and only to the internal palpebral ligament at its division. It is a strong, white, glistening membrane at its upper and outer part, but ill developed elsewhere. In the lower lid it connects the lower border of the tarsus with the orbital rim of the malar and superior maxillary bones. To the upper and inner side above the tendo oculi it is pierced by the fronto-nasal termination of the ophthalmic artery, also by its palpebral branch or branches, and by the vein of communication between the facial and superior ophthalmic veins. The supra-trochlear nerve and supra-orbital vessels can scarcely be said to do so, as they run in bony grooves or foramina and pass out above the membrane. To the outer side the palpebral branches of the lachrymal artery pierce it. The *Meibomian glands* number from thirty to forty in the upper lid, and from twenty to thirty in the lower. They are embedded in the under surface of each tarsus, and are arranged in linear series parallel to the surface. In structure they exactly resemble sebaceous glands. Each consists of a long excretory duct which extends the greater length of the tarsus, and into which several caecal appendages open. The duct is lined with stratified epithelium; the alveoli consist of a basement membrane lined by two or three layers of glandular spheroidal cells with marked reticulated protoplasm, and a large nucleus with one, two, or more nucleoli. Their excretory ducts open on the free borders of the lids. Between these ducts and the cilia is a layer of muscular tissue, which is an offshoot of the orbicularis palpebrarum—the *musculus ciliaris Riolani*; a few of the muscular fibres run along between the ducts and the conjunctiva. The *gland of Waldeyer* is a small racemose gland embedded in the hinder part of the tarsus, lined by short cuboidal cells, and secretes mucus; the ducts, two or three in number, open on the conjunctiva. The *tendon of the levator palpebræ*, upon reaching the lid, broadens out and appears to split into two planes—the anterior, or upper, is *fibrous*; and the true tendon, the posterior or lower, *muscular*. The former blends with the aponeurosis, and is attached to the anterior surface of the tarsus, to the skin, and sends various expansions between the muscular fasciculi of the sphincter orbicularis muscle. The latter is the *muscle of Müller*, and consists of unstripped muscular fibres which have an anterior attachment to the superior border of the tarsus. A similar layer of unstripped muscular fibres, also first described by Müller, exists in the lower eyelid, and is attached posteriorly to the under surface of the inferior rectus tendon and anteriorly to the inferior margin of the tarsus.

The *palpebral conjunctiva* lines the posterior surface of each lid ; it is continuous at the free margin with the skin, and extends along the canaliculi as far as the lachrymal sac. It is closely adherent to the subjacent tarsus, and, being a smooth secreting membrane, glides easily over the eyeball. Its minute structure consists of stratified epithelium and a little subepithelial connective tissue which supports vertically arranged vessels derived mainly from the superior tarsal arch, a few lymphatic vessels, and a fine plexus of nerve-fibres which renders it a highly sensitive membrane. The *palpebral arteries* proceed from the ophthalmic artery ; immediately before its final division the ophthalmic artery gives off two palpebral branches, either by a common trunk or separately, which pierce the palpebral aponeurosis directly above the tensor tarsi muscle ; thus each lid receives a palpebral artery at the inner canthus, which, dividing into two, forms by anastomosing with palpebral branches from the lachrymal artery a superior and an inferior tarsal arch in each lid. In the upper lid the superior arch lies immediately beneath the uppermost fibres of the levator palpebræ tendon, and the inferior arch between the ciliary follicles and the tarsus ; from these arches the whole lid is supplied. The sensory nerves are derived from the fifth pair. The orbicularis muscle is supplied by the facial, the levator palpebræ by the third nerve, and the muscle of Müller by the sympathetic.

The mechanism concerned in the movements of the eyelids is one of great pathological and physiological interest. The normal position of the eyelids is such that, when the eyes are open and looking straight forward, the corneæ are exposed to view except at their upper parts. This position is relatively nearly the same when the eyes are directed either upwards or downwards, the lids thus moving with the globes. The eyelids are opened by the action of the levator palpebræ, which is supplied by the third nerve. They are closed by the relaxation of this muscle, and by the contraction of the sphincter orbicularis, which is supplied by the facial nerve. The upward movement of the upper lid is effected by the contraction of the levator palpebræ, acting probably in association with the rectus superior and obliquus inferior ; and the lower lid is lifted up by means of its connection with the upper at the canthi. The lower lid is probably depressed by means of the relaxation of the levator palpebræ, and the contraction of the rectus inferior, which acts upon Tenon's capsule, with which the inferior tarsus is connected. The upper lid is thought to be pulled downwards by the lower through its attachment at the canthi. There is, besides, slight lateral movement of the eyelids when the eyeball is turned inwards or outwards, owing to expansions

from the sheaths of the internal and external recti to the conjunctiva and eyelids. The action of the muscle of Müller is involuntary: it reopens the palpebral fissure at each involuntary nictitation (blinking).

Abnormal associated movements of the eyelids.—Though the levator palpebræ is normally supplied by fibres coming from the third nerve nucleus, there is reason to believe that it may have an abnormal innervation from the fifth nerve nucleus. Many instances have now been recorded in which certain movements of the lower jaw are associated with an upward movement of the upper lid. In these cases there is usually partial congenital ptosis of one upper lid. The movements of the jaw which are attended with the associated movement of the eyelid vary: in one group of cases the lid is raised when the jaw is depressed, and also when moved to the opposite side, while in another group lateral movement produces no action of the lid, and in a third group simple depression has no effect, the associated movement being brought out only by the action of the external pterygoid of the same side.

INFLAMMATORY AFFECTIONS OF THE EYELIDS.

Blepharitis (ophthalmia tarsi, tinea tarsi, sycosis tarsi) is a chronic inflammation of the free edges of the eyelids.

Symptoms and varieties.—There are numerous degrees of the affection, varying from simple hyperæmia to severe ulceration. It begins with painful sensation of pricking and burning in the eyelids, which is increased on exposure to bright light or to cold winds. There is an increase of the glandular secretion, and the eyelids are found red and sticking together in the mornings by gummy exudation. The patient experiences inability to do prolonged eye-work. In the simplest form of blepharitis—*congestion of the lid-margins*—there are only slight redness and swelling, no marked anatomical lesion. It is usually seen in blondes with ill-developed eyelashes, and causes more distress from the disfigurement it produces than from the local discomfort. An exaggerated condition would be attended with desquamation—*blepharitis squamosa*—resembling, indeed, *eczema squamosum*. In subjects with strongly developed and thickly set eyelashes, usually brunettes, we find the glandular structures also more fully organised and taking a prominent part in the disease;

scales of inspissated sebum adhere around the exit of the lashes, much redness and swelling ensue, and the lashes are shed. This variety is an eczema seborrhoicum, and might aptly be called *blepharitis seborrhoica*. If unrelieved, yellow, honey-like crusts appear, an evidence of ulceration. There is destruction of tissue around the exit of the lashes, and altered secretion from the glands. The yellow incrustations consist of serum, altered follicular secretion, and pus. Upon their removal the margin of the lid is seen to have a 'pitted' or 'worm-eaten' appearance. In this aggravated form, *blepharitis ulcerosa* as it is called, the symptoms are more severe. There is a smarting, pricking, or burning sensation; the lachrymation is often profuse, and photophobia may exist. The lids each morning are so glued together that there is the greatest difficulty in cleansing and opening them. In another variety of blepharitis pustules form along the length of the lid like a row of styes—*blepharitis pustulosa*.

Complications and sequelæ.—If neglected, the ulceration may become deep and severe, and so destroy the glandular elements. The fatty secretion is thus suppressed, the tears overflow and cause irritation of the surrounding skin, which often becomes the seat of chronic eczema. The edges of the lids become permanently thickened, the eyelashes fall out and are replaced by others distorted and stunted in growth (trichiasis), or the lids are deprived of lashes (lippitudo), or misplaced, the margin of the upper is inverted (entropion) and causes pannus, the lower everted (ectropion) with epiphora as a result; and the palpebral fissure may eventually become considerably reduced (blepharo-phimosis), with difficulty of closure, and the cornea, as a consequence, exposed and frequently inflamed, or even ulcerated.

Etiology.—Simple congestion and slight squamous blepharitis are due, in most cases, to defective sebaceous secretion associated with slight conjunctival catarrh. Many of the worst forms of blepharitis start during an attack of catarrhal, phlyctenular, or granular ophthalmia. Very few, if any, can be disassociated with conjunctivitis. Struma, the exanthemata (especially measles), uncleanly habits, bad hygiene, lachrymal obstruction, and errors of refraction predispose to

the complaint. Its actual cause is probably parasitic in most cases, not necessarily the same micro-organism in each. Unna has described more than one organism in eczema seborrhoicum. The staphylococcus pyogenes aureus is found in the pustular variety. Though sometimes called *sycosis tarsi*, the trichophyton tonsurans, so far as I am aware, has never been seen in this affection.

Treatment and prognosis.—It is difficult to describe tersely and in detail the full management of blepharitis, so much depends upon the kind, the intensity, and the stage of the disease, also upon the age, the constitution, and the class of the patient. It is, in almost all instances, an expression of debility, so treatment must be hygienic, dietetic, and therapeutic, constitutionally and locally. If secondary to any of the forms of ophthalmia, to lachrymal obstruction, or to ectropion, such causes must be dealt with. (Cleanliness during an attack of ophthalmia is a most valuable preventive measure.) Healthy exercise, regular habits, tonic medicines, and the avoidance of stimulants are points to be remembered in constitutional treatment. Locally, congestive blepharitis requires bland preparations, as boric acid ointment, sweet almond or olive oil, cold cream, or weak ichthyol ointment. If epithelial scales are present, a lotion should be used before the oil or ointment is applied, such as a lotion of boric acid (F. 28) or subacetate of lead (F. 32). In b. seborrhoica and b. ulcerosa a more stimulating plan is requisite. It is advisable for the surgeon to remove the scales and crusts himself and estimate the damage. If there is no ulceration, a lotion of the carbonate, or bicarbonate, of soda (F. 34), or potash, should be prescribed, as well as a stimulating ointment of one of the mercurial salts, the yellow (F. 39) or red oxide (F. 42), the nitrate (F. 41), or white precipitate (F. 40). The lotion must be used warm, as a solvent to remove the crusts, every morning and evening, and the ointment applied immediately afterwards. Ulceration having ensued, it is advisable to cut the lashes short and paint the ulcerated surface either with a solution of nitrate of silver (F. 1), or with the mitigated stick (F. 7), and repeat it if necessary every two or three days. In b. pustulosa each pustule should be punctured and a perchloride

of mercury lotion (F. 31) used three times daily, and a simple ointment applied at night.

If the lower punctum is everted the canaliculus must be slit up, so that the tears may enter the sac, as constant epiphora is apt to cause eczema on the face and lower eyelid and increase the ectropion, with the establishment of a vicious circle. In all cases the refraction should be carefully worked out under atropine, and the correction ordered for constant use. If photophobia is a troublesome symptom neutral-tinted goggles with gauze sides should be worn till the acute symptoms have passed off. The patient should avoid fatigue of the eyes, cold winds, and smoky and dusty atmospheres.

Where there is much thickening of the lid-margin a complete cure cannot be hoped for, nor can the eyelashes be restored if the follicles are destroyed. Congestive blepharitis is very prone to recur, whereas the seborrhœic form in its early stage is not only amenable to treatment, but is often quickly and permanently cured. Ulceration is invariably followed by trichiasis or worse complications. The more chronic the affection the more difficult it is to cure, and that occurring in strumous subjects is usually the most obstinate.

Phtheiriasis or pediculosis is a parasitic affection which may involve the eyelids. The parasite which infests this region is the *pediculus pubis*; it will be found close to the root of an eyelash. The cilia support the nits. Low life, filthy habits, and uncleanness predispose to pediculosis. Women and children are more often the sufferers than men. The condition is readily diagnosed: numerous black granules, the ova, are visible near the exit of the lashes; with a lens the movements of the parasite leave no doubt as to the nature of the disease. There is intolerable itching, for which the patient seeks advice. The complete absence, as a rule, of inflammatory signs is in itself a sufficient distinction from marginal blepharitis. The *treatment* consists in cleanly habits, and the use of a mercurial lotion (F. 31) and a mercurial ointment (F. 41); the lids being bathed several times a day with the lotion, and the ointment applied immediately after each wash.

Edema of the eyelid is hardly an affection *per se*, but it is a symptom so often met with, and causing so much alarm to

the patient, as to deserve special notice. It may be the result of a superficial affection such as hordeolum, abscess of the eyelid or lachrymal sac, erysipelas, periostitis of the edge of the orbit, injury, or catarrhal or purulent ophthalmia. It is often noticed in the subjects of chronic nephritis on awaking from sleep, when it occurs on the side which has been lowest during the recumbent posture. It may be caused by more deeply seated troubles, such as panophthalmitis, orbital cellulitis, thrombosis of the cavernous sinus, tumour of the orbit.

Hordeolum (stye) is a circumscribed inflammation at the root of an eyelash involving its sebaceous glands.

Symptoms.—It begins as a circumscribed red patch; redness and swelling soon extend to the neighbouring parts, sometimes to an alarming extent. Pain is occasionally very severe. At the end of three or four days a yellowish point appears at the centre of the swelling, generally around the base of one of the lashes; this indicates that suppuration has taken place, and that the abscess will point externally. Several of these styes may occur at the same time, or there may be successive crops of them.

A hordeolum occurring near the inner canthus may closely simulate abscess of the sac. In such a case the sac and nasal duct should be syringed with boracic lotion or water, through the lower canaliculus. If the fluid flows freely into the nose, it is evident that there is no obstruction of the sac or duct.

Etiology and pathology.—A stye is a small boil or furuncle on the margin of the eyelid. It is an inflammation of the sebaceous glands opening into the follicles of an eyelash, also of the cellular tissue around them. This adenitis and peri-adenitis occur during a phase of increased glandular activity, at that time of life when acne spots are prone to appear, at that age when the functional activity of the sexual organs has just commenced. During this period of physiological hyperæmia it is easy to conceive how readily they may be infected by micro-organisms. Staphylococci are always found in the pus removed from a stye. Any cause of conjunctivitis, as hypermetropia, cold winds, dust, smoke, and the like, may predispose to styes. No age is exempt, for at any time the glands may be infected. A crop of styes may occur

at the same time or rapidly in succession, from local contagion. They recur from predisposition. Constipation predisposes more than any cause to a recurrence of styes; menstrual irregularities also are believed to do the same.

Treatment must be constitutional as well as local. The general health should be improved by exercise in the open air and the administration of good food, and tonic medicines, such as iron and quinine, or bark and ammonia. Great benefit is often derived from the internal administration of small doses of sulphide of calcium (gr. $\frac{1}{2}$) in the form of a pill immediately after meals three times daily. They should be continued for at least a week or ten days after the disappearance of the styes. It is necessary to see that the bowels act regularly by administering laxatives, one of the best being castor oil; or some aperient mineral water. Over-use of the eyes should be avoided. Locally the pain will be much relieved by frequent fomentations of belladonna or boric acid lotion several times a day. When pointing has commenced, the cure is accelerated and the pain relieved by a puncture with a broad needle or a cataract knife, to give exit to the pus and relieve tension.

TUMOURS OF THE EYELIDS.

Chalazion (Meibomian cyst, tarsal tumour) is a small growth situated in the substance of the tarsus.

Symptoms and clinical features.—The tumour is more commonly situated in the upper lid than in the lower; several may occur at the same time. They vary in size, their diameter ranging from 3 mm. to 10 mm. The most constant symptom complained of is the feeling of heaviness or a weight in the eyelid. The tumour is generally hard and spherical, fixed to the tarsus, but not to the skin. On everting the eyelid, a bluish discoloration is observed; this is due to thinning of the tissues beneath, and corresponds to the position of the tumour. It develops slowly, and may cause no inconvenience for several months; but, if left alone, it often inflames, and sometimes suppurates, pointing generally through the conjunctiva, but occasionally externally. In this way it may

finally disappear by contraction. As a rule, however, this spontaneous rupture is followed by the protrusion of masses of granulation-tissue on to the conjunctival surface, which are apt to cause irritation, and may continue for months before disappearing. Chalazion is sometimes difficult to distinguish from hordeolum. In making the diagnosis, it must be borne in mind that hordeolum is always an acute inflammation extending over a short but definite time, and terminating either in resolution or in suppuration; that it is always situated at the edge of the eyelid in connection with the gland of a hair-follicle. Chalazion, on the other hand, is a chronic affection, extending over months or years. It generally begins and continues for some time without marked signs of inflammation; it usually occupies the surface rather than the edge of the eyelid; its presence may be made at once evident by everting the lid, when the conjunctiva corresponding to its position will show the change mentioned above; and, finally, it tends to the formation of granulation-tissue.

Etiology and pathology.—At present little is known of their origin. The former view that they were retention cysts is untenable. They never contain sebaceous matter, have no cyst-wall, and frequently suppurate. The contents are found to be either gelatinous or purulent. The gelatinous material, examined microscopically, is seen to be composed of numerous round cells, some with epithelial characters, others not; intermingled with them are some large multinucleated cells. Tangl has expressed an opinion that they are tubercular, and in some cases he has found the tubercle bacillus. Other observers have failed to do so, nor has experimental inoculation into the anterior chamber of rabbits' eyes given any but negative results. A special chalazion-bacillus has been described by Deyl.

Treatment.—Very small Meibomian cysts, especially when they are not causing inconvenience or disfigurement, are best left alone. Large cysts must be removed by surgical means, both on account of the deformity they produce and the irritation they are likely to set up in the eye as well as the eyelid.

Operation for chalazion.—When situated in the upper lid, this must be everted, and a vertical incision, through the conjunctiva,

made deeply into the cyst, extending from behind forwards, by means of a pointed scalpel or Beer's knife. After the fluid contents have escaped, the remaining thick gelatinous-looking granulation-tissue can be removed by a small curette. If its contents are not thoroughly removed, the tumour is apt to recur. The remaining part of the cyst is sometimes thick at first, and the cavity often fills with blood immediately after the operation; but this soon shrinks, and the blood becomes absorbed in about a week. When the tumour occupies the lower lid it is not so easily removed, owing to the feeble resistance of the tarsus. In this case the eyelid is best controlled by Griffith's chalazion forceps (fig. 2) and the cyst incised on its

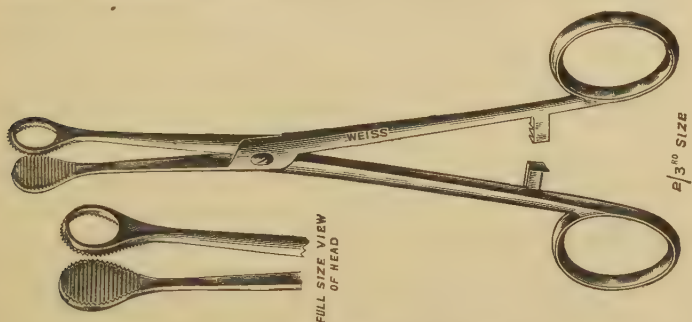


FIG. 2.—Griffith's Chalazion Forceps.

conjunctival aspect through the fenestrated blade. The contents immediately extrude, and the cavity should then be well curetted. If the outer wall of the cyst be incised, subcutaneous hæmorrhage will cause a black eye.

Herpes ophthalmicus (*Herpes zoster frontalis*, zona) is a form of herpes zoster occurring along the course of one or more branches of the ophthalmic division of the fifth cranial nerve. It is characterised by an eruption of vesicles upon an inflamed area extending from the brow of the affected side to as far back on the scalp as the lambdoid suture, and attended with considerable heat, itching, and neuralgia. If the nasal nerve is also involved, vesicles will appear on the same side of the nose. It is an affection more commonly met with in elderly debilitated subjects, but has been seen in children. Premonitory symptoms in the form of local tenderness and pain are usually complained of. Eventually the skin becomes red and swollen, and the herpetic vesicles appear in the form of

groups, which, lasting for only a few days, coalesce, and their contents dry up, leaving an irregular patchwork of brown scabs along the course of the nerve; these come away in scales, and a permanent scarring remains indicative of what has taken place.

Intractable neuralgia and ocular lesions are the two important *sequelæ* to be feared.

The only trouble for which this affection is liable to be mistaken is erysipelas; the characteristic vesicles occurring along the course of a nerve, its strict asymmetry, and the associated hemicrania are sufficient points to lead to a correct *diagnosis*.

Herpes corneæ may also occur while *herpes ophthalmicus* is at its height or as it subsides. According to Hutchinson, the eye hardly ever suffers much except when the nasal branch is affected. Vesicles occurring on the side and tip of the nose are sufficient to cause one to apprehend ocular troubles.

Treatment must be local and constitutional. Sedative lotions, as lead and opium, belladonna, and cocaine, will be most beneficial over the affected area of the skin. Internally, tonics must be given—quinine and nux vomica, or ammonia and bark. The neuralgic pains will be best relieved by the administration of chloral and bromide of potassium, and by the application of the galvanic current over the area affected.

Chalky concretions are sometimes seen in the so-called 'gouty' conjunctivitis of elderly people. They consist of phosphate of lime in the Meibomian sacculæ. As, sooner or later, they project out from their bed and cause considerable pain by scratching the cornea, their removal is necessary. This is easily accomplished with a cataract needle.

Translucent marginal cysts are occasionally seen on the margin of the lid near the inner canthus, caused by the blocking of one of the ducts of the glands of Moll; the gland becomes distended with retained watery secretion. They are readily cured by the removal of their anterior wall. If only punctured with a needle they will recur.

Dermoid cysts arising in connection with the foetal orbito-nasal fissure may appear in order of frequency at the outer angle of the orbit, at the inner angle of the orbit, in the upper eyelid, and over the line of the nasal duct.

Clinical features. Over the external angular process of the frontal bone, near the tail of the brow, is certainly the most common site. It is congenital and tends to grow slowly, but rarely exceeds the size of a cherry. The tumour is spherical, movable, and elastic. The skin, raised, appears normal and freely movable over it. On dissection the cyst is found beneath the orbicularis and attached by a pedicle to the periosteum, often being received into a shallow saucer-like depression in the bone. Rarely, in this position, do they extend into the orbital cavity, whereas to the inner side they invariably do, and some difficulty may be experienced in removing them *in toto*. Moreover, dermoids here may be so far back in the orbit that they constitute retrobulbar tumours and cause proptosis; the diagnosis then becomes difficult. The tumour in this site causes increased breadth to the bridge of the nose, with a definite swelling immediately below the orbital margin and above the inner canthus. Sometimes its pedicle may extend into the cranial cavity and be attached to the dura mater, in which case a pulsation may be transmitted to the swelling, and the diagnosis from meningocele (see Orbital Tumours) rendered anything but easy. In the upper eyelid it rarely has any bony attachment. Dermoids are not seen in the lower eyelid, but cysts in connection with ill-developed eyes (microphthalmos) may extend into it. On exposing a dermoid cyst it appears white if its contents are sebaceous, or plum-coloured if, as in some instances, it contains clear, honeylike fluid. It rarely causes more inconvenience than the deformity. There is seldom any difficulty in distinguishing it from a cavernous angioma, the latter being lobulated, reducible, and possessing, in most instances, a skin with a nævoid stain covering it.

On microscopic examination its wall is seen to resemble the skin and its appendages, such as epidermal epithelium, hair-follicles with their sebaceous glands and condensed connective tissue, which forms the outer part of the wall and blends with the surrounding tissue, in which it is embedded. The contents resemble either sebaceous secretion or honey, in which hairs, cholesterolin crystals, and epithelial cells are found microscopically.

The *treatment* consists in excision. A good large incision must be made over the tumour, which should then be well cleared from the surrounding tissues. This should if possible be done without rupturing the tumour, otherwise the contents immediately escape, and the thin walls are afterwards difficult to remove in their entirety.

Congenital Cysts.—Besides dermoid cysts, other congenital cysts are on rare occasions met with in the lids. These cysts, unlike dermoids, occur in the lower eyelid, are always associated with microphthalmos, and are connected by a pedicle, often tubular, with the ill-developed eyeball. The cyst consists of an outer fibrous wall and an inner nuclear membrane resembling the foetal retina.

Nævi occur in the eyelids. They may be small and limited to the skin—capillary angiomata; or subcutaneous and diffuse, often extending, like dermoid cysts, some distance into the orbit—cavernous angiomata. The former are slightly raised, bright red, and easily disappear under pressure; they are rarely larger than a millet seed, and are not congenital. The latter, invariably congenital, often extend into the upper eyelid from the upper and inner part of the orbit. The skin of the lid may show a port-wine stain over the tumour. These growths swell to a surprising extent when the child struggles and cries. If the skin has no capillary staining, it appears purple and lobulated and the tumour yields on pressure.

Treatment of nævi in this region must be such that no harm results from cicatrisation. For the strictly cutaneous angiomata electrolysis is an ideal method, and one or two sittings will suffice to destroy it without any scar; if a battery is not at hand, ethylate of sodium may be used with success. Cavernous angiomata, on the other hand, often prove most troublesome, and electrolysis may be of no avail. In such circumstances excision is the only obvious course to pursue, bearing in mind that the hæmorrhage is sure to be severe and that infants and young children bear loss of blood badly. If this operation is decided upon, the incision should, if possible, be made in healthy (not nævoid) skin, and the growth separated as much as possible from surrounding tissues by a tenotomy hook and a pair of forceps. A ligature is

placed firmly round its pedicle and the bulk of the vessels excised.

The operation of electrolysis.—This operation may be carried out in two ways, either by attaching a needle to each electrode of a constant current battery, or by placing a metal pad at the nape of the neck in connection with the positive electrode and utilising only one needle attached to the kathode. The former causes less shock, and is preferable if a general anæsthetic is refused. The destruction of the tissue is said to be brought about by saponification. A decomposition of the inorganic salts occurs, and the alkalis liberated at the negative pole cause, from their affinity for oxygen, a liberation of hydrogen at this pole; a soft eschar or coagulum is formed at the site of the kathode, whereas at the anode or positive pole there is developed a hard black eschar to which the needle is adherent; for this reason the negative needle, which is easily withdrawn, is preferred for making the punctures. In using two needles it is obviously incorrect to allow their points to keep in contact. It is advisable to have the battery connected to a galvanometer, so that the strength of current can be registered; it should be about 50 milliamperes. Small nævi, limited to the skin and capillary in nature, are easily destroyed by one or two applications, but the large efflorescent angiomas often seem to be aggravated by electrolysis and to grow more vigorously.

Xanthelasma palpebrarum (xanthoma) is characterised by the presence of yellow flat patches in the skin of the eyelids. The upper lid is usually attacked, but both may be simultaneously affected. They first appear above the inner canthus, and tend to spread outwards parallel to the edges of the lids or unite around the canthus. Xanthoma occurs more frequently on the left side and in women past middle age. It must not be confused with *xanthoma multiplex*, which occurs in children and is scattered as a tubercular eruption on the trunk and limbs. Hutchinson considers xanthoma palpebrarum to be the outcome of recurrent attacks of headache and pigmentation of the eyelids seen in some women. Panas has shown it to consist of changes in the cutis vera; coarse granular cells grouped together in places, and believed to be a fatty degeneration of endothelium. Defective nutrition with dilatation and thrombosis of lymph fissures is the pathology according to Knies. Sebaceous cysts occasionally develop in

these patches. Their presence causes no pain or inconvenience ; but when numerous and of considerable size they are cosmetically objectionable, and the patient may desire to have them removed. This can be easily done by raising them with forceps and using a pair of curved scissors. A suture may be required, but no scar is perceptible after the operation.

Rodent ulcer (dermal carcinoma) is the most common malignant growth attacking the eyelid.

Symptoms and clinical features.—It seldom appears before the age of forty. It most commonly attacks the skin near and below the inner canthus, but it may occur in other parts of the eyelids. It first appears as a small hard nodule, which the patient describes as a ‘pimple ;’ this sooner or later becomes covered with a squamous incrustation, beneath which the skin is found to be excoriated. At this stage it causes but little inconvenience ; the patient is in the habit of wiping away the scab from time to time, but finding that it does not heal, he presents himself to the surgeon, and it is at this period of the disease that we generally see it. It now presents a dry brown scab, which consists of inspissated blood ; beneath this is an ulcerated surface, which at first may be little more than an excoriation, and may appear to heal up for a time, but soon breaks out again, and becomes deeper, with hard, nodulated, everted edges and purulent discharge. It may remain indolent, or only occasionally irritable, for months and even years, without making visible progress either in surface or depth ; but sooner or later it will take on a rapid action, destroying not only the skin, but the deeper parts of the eyelids, the connective tissues of the orbit, the cornea and globe of the eye, and finally the bones of the face. Rodent ulcer is regarded as the mildest expression of a malignant disease—chiefly because of the long indolent stage, during which there is no pain and no infiltration of the neighbouring lymphatic glands—but as soon as the active stage has commenced, and the deeper tissues have become affected, the pain grows intense, and the destruction of tissue is so rapid that the term ‘mildness’ is no longer applicable.

Histology.—Rodent ulcer under the microscope resembles an alveolar carcinoma. Cells in great numbers are seen closely packed in intercommunicating spaces. The cells are small and

often appear spindle-shaped. They may be grouped around a Meibomian duct, and in some cases the tumour seems to have originated in the surrounding acini. The epidermis in an early stage is unaffected, but later its cells undergo proliferation, and then 'cell-nests' may be seen."

Epithelioma (epidermal carcinoma) is less frequently met with in the eyelids than rodent ulcer. It infects the lymphatic glands, and hence is more malignant.

Epithelioma varies clinically from rodent ulcer in that there is more outgrowth, it infects the neighbouring glands, it never tends to cicatrise, and its seat of election is not so constantly near the inner canthus. In that it usually starts on the lid-margin it upholds the usual feature of these growths, viz. growing at the junction of skin and mucous membrane. It often resembles a large sprouting molluscum contagiosum. When it has ulcerated, which it does early, the margin of the ulcer is raised, rounded off, pale pink with semi-translucent appearance, and of cartilaginous consistence; a condition more constant in epitheliomatous than rodent ulcers. The rough papillary surface in the centre easily bleeds and forms a blood scab. The lymphatic glands are involved late.

Its histology differs immaterially from epithelioma in other parts. The large size of the cells, the presence of epithelial pearls ('cell-nests') and 'prickle' cells, and the thickening and inroads of the surface epithelium are sufficient characteristics to distinguish it from rodent cancer. Cell-inclusion and irregular mitosis are also more easily noticed.

The *diagnosis* of rodent ulcer from tertiary syphilitic ulcer is sometimes difficult. As a rule, however, the age and history of the patient, the presence of collateral evidence of syphilis, and, locally, the sharply defined edges of the ulcer, the duration of its existence, and its rapid subsidence under anti-syphilitic remedies, easily clear up the diagnosis. It is to be recognised from a chancre in the absence of glandular enlargement, by its slow growth, and, if any doubt exist, by its failing to yield to mercury. The advent of the secondary manifestations of syphilis associated with enlargement of the pre-auricular gland would exclude epithelioma. Moreover, the glands are not infected early in epithelioma of the eyelid.

The *treatment* of rodent ulcer and epithelioma consists in the effectual removal of all the diseased tissue. This may be done in various ways, either by the knife, the thermal cautery, or by scraping with a steel scoop.

In an early stage, while the ulcer is movable—*i.e.* not anchored to the bone—excision may result in a permanent cure. Before excising the growth the eyelids should be pared along their margins and united (tarsorrhaphy), as a guard against cicatricial ectropion. A zone of healthy skin and tissue must be removed with the growth, and if the ulcer is found adherent to the anterior wall of the lachrymal sac, this cavity must be opened, its anterior wall removed, and its lining membrane scraped away with a curette so as to completely destroy it. A V-shaped excision is sometimes necessary for growths occurring in the middle of the eyelid; in such cases a tenotomy of the tendo oculi will assist materially in bringing the edges of the wound together, and will relieve tension on the stitches. When both eyelids are involved, necessitating their removal, the eyeball, even though it be perfectly healthy, must be excised, a sacrifice of which the patient should fully understand prior to being anæsthetised. If operation be refused, or the area ulcerated so extensive as to contraindicate surgical interference, much relief may be given the patient by keeping the part thoroughly clean with an antiseptic lotion, and painting on cocaine if the pain is severe.

Patients will often rather suffer severe pain from caustics than submit to an operation. In such circumstances the growth must be destroyed by the use of chloride of zinc paste, chromic acid, or chlorate of potash, after an application of cocaine.

The X-ray treatment of rodent ulcer often proves most efficient in checking the progress of the disease.

Papillomata (warts) are occasionally found on the edge of the eyelid near the ciliary margin. They should be snipped off with curved scissors, care being taken to cut well below their bases. **Horns** are also sometimes seen, and should be treated in a similar manner.

Molluscum contagiosum is an epithelial affection of the pilosebaceous glands, and is most commonly seen on the eyelids and face. It has been proved to be contagious.

Clinical and pathological features.—One, two, or more hemispherical umbilicated prominences of a white waxy appearance present themselves on one or both eyelids of a child.

They vary in size from a pin's head to a large-sized pea. No pain nor inflammation as a rule attends them. They take months in growing to any appreciable size, and occasionally inflame and necrose; extrusion with a spontaneous cure is the result. In fact, if left alone they invariably disappear spontaneously. Their histological characters are essentially epithelial. They consist of numerous follicles closely packed together, which open chiefly at the central umbilication. The base is lobulated, and situated 1 mm. or 2 mm. below the level of the corium. The skin over the larger growths is stretched and devoid of papillæ. Each follicle has a basement membrane which supports a single layer of columnar cells; then two or three rows of polygonal cells, a row of granular cells resembling the stratum granulosum of the cuticle; lastly, and most internally, one, two, or more rows of clear spheroidal cells, called 'molluscum corpuscles,' which stain yellow with picro-carmin. It is asserted by some pathologists that these corpuscles are coccidia, by others that they are merely altered epithelial cells.

Treatment.—Each tumour must be transfixed through its base with a small scalpel, and divided; its contents can then be evacuated either by squeezing between the thumb-nails or with forceps.

Sarcoma as a primary growth in the eyelid is an extremely rare affection. A definite tumour forms, hemispherical in shape and not unlike a chalazion. It rapidly exceeds the dimensions of a cyst, and the skin, taut over it, becomes adherent, but does not ulcerate early. A sarcoma in this region is as often pigmented as not, and may take its origin in a pigmented mole. It usually takes the form of a round- or spindle-celled growth. Free removal is the only treatment likely to be attended with success.

Lipoma, Fibro-adenoma, Lymphangioma, and Molluscum fibrosum of the eyelid are very rare. They present the same characters here as in other regions.

Neuroma.—There are about twelve cases on record of a plexiform neuroma of the eyelid and orbit. The tumour consists of more or less degenerated nerve-fibres packed together by whorls of dense connective tissue.

Indurated chancre sometimes occurs on the eyelids, often

at the inner canthus. It is usually accompanied by much swelling. The pre-auricular gland becomes enlarged and indurated. It may be mistaken for a tertiary syphilitic ulcer, a rodent ulcer, a large molluscum, or a tubercular ulcer. The presence of swelling of the pre-auricular gland excludes the first three lesions; the last is more difficult to exclude, but the existence of induration accompanied with much inflammatory reaction will indicate a chancre.

Gummata occasionally occur in the eyelids, their seat of election being usually at the outer part of the upper lid. The induration is sometimes accompanied by swelling of the surrounding tissues of the lid, and more or less redness. Besides local gummata, the tarsus may be the seat of a diffuse syphilitic inflammation—*interstitial tarsitis*. The history, gummata elsewhere, and the action of iodide of potassium will assist in making a diagnosis.

Tertiary syphilitic ulcers also occur on the eyelids. When more than one ulcer is present they are easily recognised, but when occurring singly, with indurated edges and of slow increase, they are frequently difficult to distinguish from epithelioma (see p. 19).

DEFORMITIES OF THE EYELIDS.

Blepharospasm is an involuntary closure of the eyelids owing to spasm of the orbicular muscle. It may occur as a symptom of some other eye-affection, or it may exist by itself.

Symptomatic blepharospasm is likely to accompany any irritative affection of the eye such as the presence of a foreign body in the palpebral sac, abrasion or ulcer of the cornea, inversion of the eyelashes, and the various forms of conjunctival inflammation. The severity of the spasm is not, however, always in proportion to the severity of the affection which it may accompany. It is frequently intensified by any attempt to examine the cornea by separating the eyelids in a bright light. One of the most acute and obstinate forms is that which often attends phlyctenular ophthalmia in strumous children. These poor little sufferers are quite unable to open

their eyes in any but the darkest room, and will skulk away into shaded corners all day long in order to avoid the light.

The treatment of symptomatic blepharospasm consists in removing as far as possible the affection of the eye which causes it. In order to render examination of the cornea and palpebral sac more easy, a 4 per. cent. solution of cocaine hydrochlorate should first be dropped into the palpebral sac every few minutes. This drug is very efficient in overcoming the spasm, and thus facilitates the opening and eversion of the eyelids. If cocaine does not suffice for this purpose a general anæsthetic should be employed. In those cases of entropion which result from blepharospasm in subjects with small palpebral apertures, division of the outer canthus (canthoplasty) may be all that is needed.

The chief forms of blepharospasm which are not accompanied by other eye-affection are the hysterical and the convulsive.

Hysterical blepharospasm.—In young hysterical subjects it is not uncommon to find one or even both eyes suddenly closed, whilst the patient declares she is quite unable to open them. The affection can be diagnosed from ptosis by the fact that in ptosis, when the upper lid is gently raised by the finger, there is no resistance, whilst in hysterical blepharospasm this act is resisted by spasm of the orbicularis. Uncontrollable fits of crying, polyuria, and similar hysterical symptoms are often accompaniments. These cases get well of themselves after varied intervals of weeks or months.

Convulsive tic usually attacks one eye only. It consists of a series of spasms of the orbicular muscle, which vary in severity and in duration. The eyelids are observed to twitch, at first slightly, and then, after a few seconds, more severely, until the lids are finally closed and the patient quite unable to open them perhaps for several minutes. In severe cases the spasmodic twitching extends to the entire side of the face. The attacks appear to be accelerated by any excitement, such as the presence of a stranger, or the crossing of a crowded thoroughfare. The cause of these nictitations is unknown, and their treatment has not hitherto been attended with much satisfaction.

Paralysis of the orbicularis palpebrarum is characterised by inability to close the affected eyelids (*lagophthalmos*); it is usually associated with other signs of paralysis of the facial nerve, which supplies this muscle. In recent cases no marked changes can be perceived in the affected eye, but in those of long standing the eye exhibits signs of disturbance resulting from exposure. The lower part of the cornea, which, during sleep, is incompletely covered by the lower lid, becomes dry and cloudy. The lower lid falls away from the globe and forms a kind of ectropion; and the lower punctum lachrymale, being thus everted, causes a disturbance in the flow of the tears. In slight cases this epiphora is almost the only trouble complained of by the patient. This paralysis of the facial nerve may be due to a nuclear lesion, or, as is mostly the case, the lesion may be situated somewhere in the course of the nerve outside the skull, in the middle ear, in the parotid region, or in its terminal branches. Paralysis of the sixth nerve on the same side, with crossed hemiplegia, is often associated with nuclear lesions of the facial (see chapter on Paralysis). The most frequently occurring form of facial paralysis is that which is due to neuritis; it is sometimes termed 'rheumatic.' It often follows exposure of the face to severe or prolonged cold, as in driving in a cold wind or sleeping in a draught of cold air. The prognosis in such cases is usually good, although it may require some weeks or even months to effect a cure. Daily massage, fomentations, and the use of the constant and induced electrical currents are the chief points of treatment. During the treatment the eye should be kept closed at night by a light bandage. In incurable cases the exposure of the eye by ectropion may be modified by the operation of tarsorrhaphy, in which the edges of the lids are pared near the outer canthus, including the angle, and then made to unite by sutures.

Ptoxis presents itself in all stages, varying from a slight drooping to a complete falling of the upper eyelid. In slight degrees the lid covers only a little more of the corneal surface than it ought to do; but on telling the patient to look upwards it will be found that the lid does not move with the globe, but allows the cornea to glide beneath it. The patient will now

be observed to wrinkle the forehead in order to bring the fibres of the occipito-frontalis muscle into play, and so to further elevate the lid. In severe cases the whole cornea is covered by the drooping lid, and the patient can only see by lifting the lid with his finger.

Ptosis may be hysterical, congenital, or paralytic.

Hysterical ptosis is practically identical with hysterical blepharospasm (p. 23), and requires no further description.

Congenital ptosis is the result of insufficient development, or even complete absence, of the levator palpebræ muscle. It is generally bilateral and partial, and is sometimes transmitted by heredity through several generations. It is frequently associated with other congenital anomalies, one of which is the absence or defective development of the superior recti muscles. When both sides are affected, the patient cannot see well in front of him without throwing the head backwards, wrinkling the forehead, and protruding the chin, characteristic of double ptosis.

Paralytic ptosis is the result of paralysis or injury of the branch of the third nerve supplying the levator palpebræ muscle. When resulting from disease, it must be treated in the same way as other forms of paralysis of the third nerve, viz. by the internal administration of iodide of potassium, the use of the galvanic and faradic currents, and massage. The result of such treatment is often very satisfactory, the ptosis entirely disappearing, together with such other symptoms of third-nerve paralysis as may accompany it. In any case, the therapeutic treatment should be steadily persevered with for many months unless a cure has been previously effected.

Slight ptosis is sometimes caused by paralysis of Müller's muscle. This is accompanied by contraction of the pupil, diminished intra-ocular tension, enlarged retinal veins, and hypersecretion of the conjunctiva on the affected side. It is symptomatic of disease of the cervical sympathetic, Müller's muscle being supplied by this nerve; so in all cases the neck should be examined for a tumour or aneurysm, which by its pressure may cause paralysis of the cervical sympathetic cord.

Operative treatment of ptosis.—Congenital ptosis and ptosis from

injury to the levator palpebræ muscle are the only varieties which demand operative interference. Ptosis in third nerve palsy is Nature's protection against diplopia. If medicinal remedies failed to cure the paralysis, the restoration of the use of the eyelid by operation might be attended with troublesome diplopia.

1. In cases where the ptosis is only slight, and where there is evidently some remaining power in the levator muscle, the method of von Graefe may be employed with advantage. This consists in removing an elliptical portion of the skin and the orbicular muscle from the upper lid at a distance of 5 mm. from its ciliary border, and extending from one end of the lid to the other. If the skin is abundant, a wide strip of this should be removed; if scanty, a narrow strip will suffice. Care must be taken not to remove so much



FIG. 3.—Partial Congenital Ptosis.

as to prevent closure of the eye during sleep. The edges of the muscle must first be brought together by fine catgut sutures, then the edges of the skin-wound.

The aim of the operation is to assist the levator palpebræ—firstly, by weakening the orbicular muscle, which is its antagonist, and, secondly, by shortening the eyelid.

2. In cases where the levator has little or no action, an attempt must be made to establish connection between the lid and the occipito-frontalis muscle.

Pagenstecher's operation for partial ptosis is performed as follows: A strong thread is armed with a needle at each end. One needle is then introduced beneath the skin of the upper lid parallel to and near its ciliary border for about 1 mm. or 2 mm. At the point of exit the same needle is again introduced and carried beneath the skin, but over the tarsus, and again brought out about 2 cm. above the superciliary arch, and 2 mm. external to its middle line. The second needle is then introduced at the point of entry of the first needle, directed upwards, and brought out at the same point of

exit above the superciliary arch. The two threads are then tied together over a piece of drainage-tube and moderately tightened, thus forming a subcutaneous ligature, which, being tightened each day, may be allowed, if necessary, to cut its way out. By this means a subcutaneous cicatricial band is produced, which will transmit the action of the occipito-frontalis to the upper lid.

For complete ptosis.—A needle armed with a thick thread is introduced beneath the skin about 2 cm. above the superciliary edge, and 2 mm. to the outer side of its middle line. It is then guided downwards and inwards beneath the skin, and brought out about the middle of the upper lid close to its ciliary margin. The ends of the thread are then tied in a knot, and moderate tension is made. The tension is gradually increased day by day, so as to make it cut its way through the skin, by drawing it tighter. The inflammatory symptoms are comparatively slight. The scar is not extensive. One ligature generally suffices, but two may be required.

Panas's operation consists in dissecting and gliding a flap of skin from the upper lid beneath the skin of the eyebrow, and causing it

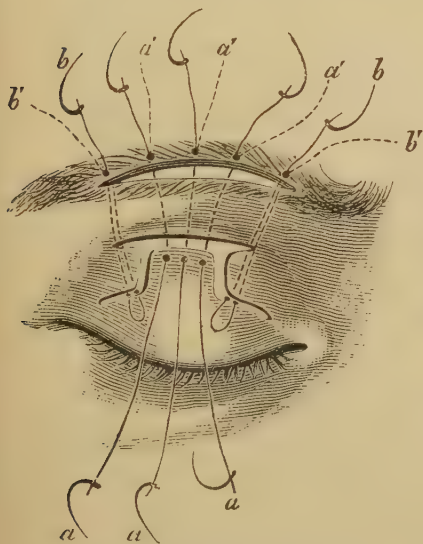


FIG. 4.—Panas's Operation.

a a', central sutures ; *b b'*, lateral sutures.

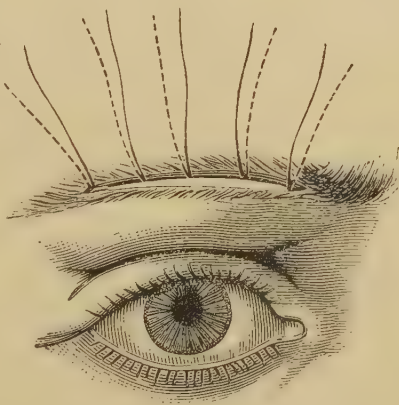


FIG. 5.—Panas's Operation (after).

to unite with the skin of the forehead immediately over the fibres of the occipito-frontalis muscle. It is performed as follows : An

incision rather more than an inch in length is made along the upper edge of the eyebrow, so deep as to expose the periosteum without wounding it. A second incision is then made about an inch in length along the fold separating the eyelid from the eyebrow (see fig. 4) just above the margin of the orbit; this must also be made to divide the orbicularis muscle and to expose the periosteum. Care must be taken not to cut below the margin of the orbit, otherwise the suspensory ligament of the lid will be divided. The bridge formed by these two incisions must now be undermined with a steel director. This being done, a curved incision is made extending to within a few millimetres of each canthus, as shown in fig. 4, dividing the orbicularis and exposing the tarsus. The flap thus marked is then dissected carefully away from the tarsus down to its ciliary border.

Fine catgut sutures, armed with a small needle at each end, are now introduced into the flap, and their proximal extremities are passed beneath the bridge of skin and eyebrow and through the skin at the upper edge of the wound above the brow; their distal ends are then also passed beneath the same archway. Now, by making slight tension upon the lid-flap by means of the sutures and by manipulation, the upper part of the lid-flap can be brought into apposition with the upper edge of the frontal wound and secured in that position by tying the sutures (see fig. 5). This operation produces excellent results, whilst the deformity is very slight.

Eversbusch's operation is sometimes employed when there is some remaining power in the levator palpebræ. It consists in the advancement of the tendinous attachment of the muscle on to the anterior surface of the tarsus, and is most useful in cases of partial congenital ptosis.

The following are the chief points of the operation: A transverse incision is made through the skin and orbicular muscle of the lid, parallel to its ciliary margin, and midway between the latter and the brow. The tendinous insertion of the levator palpebræ muscle is exposed. Through its centre, and as high up as possible, a suture, armed with a curved needle at each end, is passed so as to take up some of its fibres; both needles are then passed downwards beneath the skin and orbicular muscle to the ciliary margin, and the suture is tied tightly over a glass bead internal to the eyelashes. Two other lateral sutures are used in a similar way. The use of the beads is obviously to prevent the sutures from cutting into and damaging the margin of the lid. The eyes should be bandaged for at least a week, at the end of which time the sutures may be removed and the dressings abandoned.

C. Hess's operation.—The eyebrow being shaved, an incision is made in it following its curve and throughout its length, so as to open up the subcutaneous connective tissue. From this point the skin of the eyelid is carefully dissected from the orbicular muscle as far as its ciliary margin and throughout its length and breadth. Three silk sutures, each armed at each end with a curved needle, are introduced so as to make traction on the skin of the lid—one central and two lateral. The central suture should be entered through the skin only, and from without inwards, midway between the brow incision and the ciliary margin of the eyelid, the point of entry of the two needles being about 5 mm. apart. The ends are brought out at the brow opening. The two lateral sutures are similarly introduced. The sutures are then entered deeply under the upper margin of the incision into the substance of the frontalis muscle and brought out just above the brow. Each suture is firmly tied in a bow over a piece of drainage-tube, and, if necessary, tightened each day. The brow incision is finally closed by a continuous suture. The effect of the operation should be seen immediately; the palpebral aperture should be a little wider than normal to allow of a slight yielding. The stitches are left in for ten days. The operation is tedious, attended with considerable hæmorrhage, and there is a danger of 'buttonholing' the skin, but the result is satisfactory. The most essential feature in the operation is to obtain an extensive raw surface. It would not suffice to end the dissection at the point where the sutures are entered.

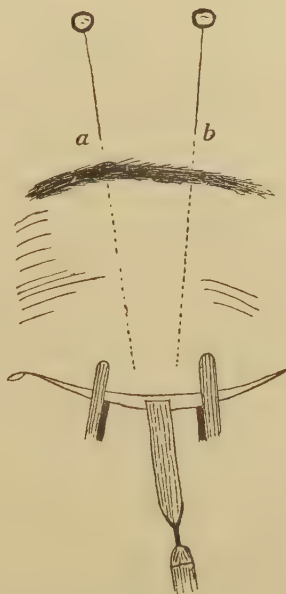


FIG. 6.—Mules's Operation.

Mules's operation (fig. 6).—Mules corrects the deformity by raising the lid with a subcutaneous wire permanently buried in the tissues. Two needles with their eyes near their points are entered above the brow at *a* and *b* and passed downwards to the lid margin

beneath the skin, and are made to emerge at the intermarginal space about a centimetre apart. Into the eyes of the needles the extremities of a piece of silver wire are threaded, previously softened in a spirit lamp, and the needles are withdrawn. A subcutaneous ligature is thus produced with a loop of wire at the lid margin; this is made to lie in a shallow bed by a slight horizontal incision. One end of the wire is now passed beneath the skin to emerge at the same aperture as the other end. The ends are twisted together till the requisite amount of elevation is acquired and then buried in the tissues. It may be necessary to tighten the ligature after a few days. Unless any severe reaction ensues, the wire should be left in permanently. The result in many instances has been good.

Trichiasis, **Distichiasis**, and **Entropion** are all modifications of the same affection of the eyelids. In *trichiasis* the eyelashes are inverted so as to rub against the surface of the globe; the number of the lashes which are thus turned in varies from one, two, or three to the whole number. Trichiasis may be congenital or acquired; the former is more often symmetrical than not, the latter is the result of inflammation or injury. In *distichiasis* there appear to be supplementary rows of cilia developed, which are incurved. It may also be congenital or acquired; if the latter, it is generally attended with more or less thickening of the free edge of the eyelid. In *entropion* there is inversion of the lid as well as of the cilia. The amount of inversion varies from a slight incurvation to complete reduplication, so that the cilia are in contact with the cul-de-sac. Entropion may be acute (spasmodic) or chronic (cicatricial). The acute form is common in the lower lid in old people from blepharospasm caused by a foreign body, inflammation, or after an operation on the eye. The chronic form is usually due to cicatrisation of the inner surface of the upper lid.

Causes.—The most common cause of all these affections is chronic granular conjunctivitis, which, having been imperfectly cured, has been followed by contraction of the conjunctival surface of the lid. Sometimes they are due to contraction of the sphincter orbicularis. They may be the result of injuries of the conjunctiva, lacerations, burns, xerosis, &c.

Treatment.—*For trichiasis and distichiasis.* When the

number of incurved cilia is small they may be removed by epilation forceps (fig. 7). Each lash should be firmly seized close to its base and pulled out steadily. They soon recur, and may be subjected to the same treatment. In case



FIG. 7.—Epilation Forceps.

of a third or fourth recurrence, some method of destroying the incurved lashes should be adopted. Various methods are employed for this purpose.

(1) *Electrolysis* has proved a simple and successful method of destroying incurved lashes. The positive pole (anode) of a constant current battery is applied to the temple on the side of the affected eye, the negative pole (kathode) is attached by an insulated handle to a sharp sewing-needle. The eyelid is fixed by means of a Snellen's clamp so as to expose the incurved lashes. The needle is now pushed well down to the root of each offending cilium, and a current of three or four milliamperes turned on. A slight bubbling of gas will be observed, and the lash will become loosened so that it can be pulled out without any difficulty. The process is a painful one, and the simple instillation of cocaine into the palpebral sac does not suffice to prevent suffering. Either cocaine must be injected subcutaneously into the affected area or a general anæsthetic used.



FIG. 8.—Lines of Incision in Arlt's Operation.

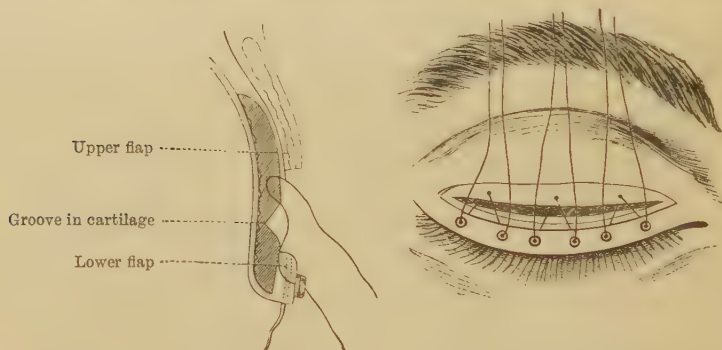
(2) *Transplantation* of the ciliary margin of the eyelid is necessary if the number of incurved eyelashes is considerable, as in distichiasis and severe cases of trichiasis.

Arlt's operation.—A small double-edged straight knife is inserted at one or other end of the eyelid between the cilia and the Meibomian ducts, and its point is made to come out through the skin about

2 mm. above the lashes. It is then made to cut its way along the whole length of the edge of the lid (see fig. 8), and thus forms a bridge of tissue containing the lashes only. A second incision is now made from the two extremities of the first, curving upwards to the extent of 3 mm. or 4 mm. This forms a semilunar flap on the upper lid which must be dissected off. The bridge of skin containing the cilia has now to be shifted upwards, and its upper edge attached by sutures to the skin of the lid, its lower edge being left free. A dry antiseptic dressing is all that is necessary.

Another method of dealing with extensive trichiasis is that of *scalping*. The whole ciliary margin of the lid is dissected away. The practice is becoming obsolete.

For entropion. (1) *When spasmodic*, the entropion may be temporarily relieved either by painting collodion on the lower eyelid or by applying a strip of adhesive plaster. By far the best method,



FIGS. 9 and 10.—The Streatfeild-Snellen Operation for Entropion.

both for congenital and spasmodic, is the excision of a strip of skin and orbicularis muscle from the whole length of the lid parallel to its margin. The width of the flap to be removed must vary according to the laxity of the tissue, which is generally great in these cases. The edges of the wound are united by sutures, and a dry dressing applied.

(2) *When chronic*, it may be treated by one of the following methods :

A. By *Arlt's method* of transplanting the ciliary border, which is the same as that just described for trichiasis.

B. By *Streatfeild's operation* for 'grooving' the tarsus. This is best described in Streatfeild's own words: 'The lid is held with

entropion forceps (fig. 11), the flat blade passed under the lid, and the ring fixed upon the skin so as to make it tense, and expose the edge of the lid. An incision with a scalpel is made of the desired length, just through the skin, along the palpebral margin, at a distance of a line or less, so as to expose, but not to divide, the roots of the lashes; and then just beyond them the incision is continued down to the cartilage (the extremities of this wound are inclined towards the edge of the lid); a second incision, farther from the palpebral margin, is made at once down to the cartilage in a similar direction to the first, and at a distance of a line or more, and joining it at both extremities; these two incisions are then continued deeply into the cartilage in an oblique direction towards each other. With a pair of forceps the strip to be excised is seized, and detached with

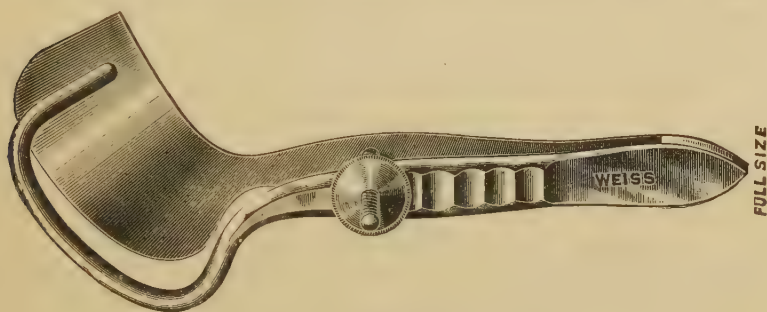


FIG. 11.—Snellen's Entropion Forceps.

the scalpel.'¹ Three sutures are then introduced as follows: A small curved needle, armed with fine silk, is passed first through the lower edge of the skin-wound, then through the upper edge of the groove in the tarsus, and the two ends tied tightly together. The upper edge of the skin-wound is thus left free, and unites very well without sutures. This operation gives excellent results. It has been slightly modified by Snellen, who makes the groove in the cartilage rather higher up, and uses a different form of suture. Three sutures are used (see figs. 9 and 10). A fine silk suture is passed through the upper edge of the groove in the tarsus for about 1 mm. It is armed at each end with a needle. These needles are then passed through the lower edge of the skin-wound, just above the cilia; their distance apart should be about 4 mm. All the sutures being similarly introduced, a glass bead is passed over the end of each, and they are all tightened together by gentle traction, and then each suture tied.

¹ *R.O.H. Reports*, vol. i. p. 125.

C. *By grafting.* This operation was first demonstrated to me by van Millingen of Constantinople, and has proved itself to be a most efficient method of relieving the cornea from the irritation of the lashes and the free edge of the upper lid in old-standing entropion. It consists in separating the free edge of the tarsus from the eyelashes by incision, and in transplanting a portion of the mucous membrane of the lower lip of the patient into the gap thus formed.

Operation.—A horn spatula is placed beneath the lid as far as the upper border of the tarsus, and is held in that position by an assistant, who endeavours to tilt its upper end forwards in such a manner that the surgeon can make tension upon the skin of the lid by dragging upon the latter with the fingers of his left hand whilst, with a sharp scalpel in his right hand, he splits the lid by an incision along its whole length just internal to the roots of the lashes. This should be about 3 mm. deep. Then, continuing to make traction with the fingers, he proceeds to free the tarsus still further by a series of small cuts until an interval of about 5 mm. is produced. This wound is now made to gape by means of three sutures, passed first through the skin of the lid close to its ciliary border, and then just below the eyebrow above—at the outer, middle, and inner portions. By now tying up the sutures moderately tightly, the wound will be found to gape in such a way as to favour the reception and adaptation of the graft from the lip. Hæmorrhage being stopped and the mucous membrane of the lip duly washed, the latter is made slightly tense by an assistant, and a long narrow strip is excised with scissors and placed in the gaping wound. In excising the graft, it must be remembered that it will shrink immediately after separation from the lip, and so should be made rather large and tapering at each end. In placing it in the wound, care should be taken to keep the mucous surface outwards so that the raw surface can come into immediate contact with that of the wound; whilst its free edges and tapering extremities should be carefully tucked into the wound, so that the latter may hold it *in situ*. Some surgeons employ fine sutures for this purpose. The surface is then covered with dressing of iodoform ointment, and bandaged. Sutures are placed in the lip-wound. The stitches are removed from the upper lid on the fourth day, by which time the grafts are usually well united; and the bandage is replaced by a shade. On the fifth or sixth day the patient can usually open the eyes quite freely, and from that time experiences great relief.

D. *Scott's operation* (fig. 12).—Many operations for entropion best do not relieve the pressure of the sharp incurved edge of the tarsus the cornea, although the trichiasis may be cured. This operation

does away with the cicatricial kink of the eyelid. After a spatula has been introduced into the upper cul-de-sac the upper lid is everted, and an incision is made along its whole length on its conjunctival aspect and about 2 mm. from its margin (fig. 12, 1). It must divide the tarsus completely in its whole thickness and from end to end. The ciliary portion of the eyelid is now taken in a pair of dressing forceps and forcibly everted. A curved needle threaded with silver wire is passed vertically downwards, entering through the centre of the cutaneous surface of the eyelid into the substance of the upper portion of the tarsus, and made to emerge in the middle of its divided

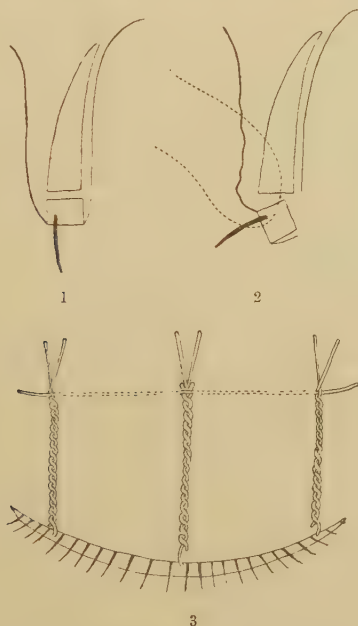


FIG. 12.—Scott's Operation for Entropion.

1. Incision in eyelid.
2. Eversion and suture.
3. Lid-sutures attached to buried eyebrow-suture.

edge; it is then made to enter the original anterior surface of the lower separated and everted portion of the tarsus, to be finally brought out on the free margin of the eyelid (fig. 12, 2). Two other sutures are similarly introduced, one on either side. The opposing ends of each wire suture are twisted together so as to form three cords. Each one is connected with a fourth, passed horizontally through the brow and drawn sufficiently tightly to evert the lower portion of the tarsus (fig. 12, 3). The reaction is slight, and the

operation may be performed under cocaine. The stitches should be removed about the sixth day. My opinion of this operation is certainly one of commendation.

Ectropion is that condition in which the eyelid is everted and its conjunctival surface exposed. It most commonly affects the lower lid. It may be *partial* or *complete*, and the eversion may cause displacement of the lower punctum lachrymale, drawing it so much away from the globe as to prevent the tears from entering it. The exposed conjunctiva is often thickened.

Causes.—Acute spasmodic ectropion, partly due to spasm of the orbicularis palpebrarum and partly to tumefaction of the conjunctiva, is sometimes met with in ophthalmia neonatorum, in which the lids become everted on the orbicularis muscle being called into action, as in crying, &c. Chronic ectropion may be caused by facial paralysis, blepharitis, chronic senile catarrh of conjunctiva, and cicatrices the result of wounds, burns, ulceration, and bone disease, and following operations for rodent ulcer, lupus, &c.

Treatment must vary with the cause of the eversion.

1. In the *acute* form, if the lids persistently revert each time they are replaced in their proper position, or if a condition allied to paraphimosis occurs, it will be necessary to divide the outer canthus. This will do away with the *point d'appui*, and also relieve any strangulation of the conjunctiva. If the ectropion is only occasional and does not immediately return after replacement, the condition is not serious and will cease directly the swollen conjunctiva subsides.

2. In the *chronic* varieties, ectropion from facial palsy must be cured by galvanism; that due to thickened conjunctiva and old blepharitis in which the punctum lachrymale is found everted, by slitting up the canaliculus into the sac and keeping it open. Some operation may be of service.

A. *Argyll Robertson's method* is well suited for cases of ectropion of the lower lid in old people, in which the conjunctiva is thickened and the tissues of the face lax. Each end of a stout ligature, armed with a needle, is passed from without inwards through the margin of the eyelid, the punctures being about a centimetre apart. In this way a loop is left externally parallel with the edge of the lid. Each

end is then thrust through the lower cul-de-sac and made to emerge upon the cheek well below the eyelid.

The operator now takes a piece of sheet-lead, shaped and moulded to resemble the normal tarsus: this he places in the conjunctival cul-de-sac, beneath the ligatures, so that, on tightening the latter, the lid is moulded to the lead, and lead and eyelid are together drawn towards the eye. A stout piece of drainage-tube is now placed beneath the external loop, and the ends of the ligature tied over it below; this prevents the skin being cut, and by its elasticity allows a certain amount of swelling to occur. After about ten days the ligatures are cut and removed, when a considerable improvement, and often a complete cure, will be found to have been effected.

B. When the edge of the lid has become elongated as well as everted, *Adam's operation* may be performed. This consists in removing a triangular wedge from the whole thickness of the lower lid (see fig. 13). The base of the triangle must be at the edge of the lid; its width may vary, according to circumstances, between 5 and 10 mm. The sides of the triangle should be from 10 to 20 mm. The edges of the wound are brought together by a fine pin, and secured by one or two sutures. This operation is often more advantageously performed near the outer canthus.

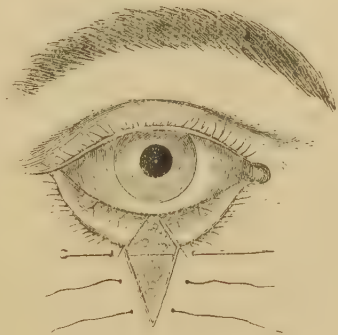


FIG. 13.—Adam's Operation for Ectropion.

When the eversion is due to contraction of neighbouring cicatrices on the face, the nature of the operation must depend upon the site and extent of the lesion, and much scope is often afforded for the exercise of ingenuity on the part of the surgeon.

In all such cases no plastic operation should be attempted until the skin of the affected part has as far as possible recovered from the injury. It sometimes requires six months or more for the hardness and thickening of the skin and subcutaneous tissue to have passed away. Blepharoplastic operations for cicatricial ectropion are many and varied, and are undertaken not only for cosmetic reasons, but for the preservation of the eyeball. In slight cases Wharton Jones's

operation will cure the eversion, but where much ectropion exists it is of no use; some method of skin grafting must be employed, such as that of Dieffenbach.

C. *Wharton Jones's operation*.—The eye is to be protected by a horn spatula placed beneath the lower lid. A V-shaped (fig. 14) incision is to be made with a small scalpel, including as far as possible the cicatricial tissue; the flap thus formed is to be dissected from the subjacent parts sufficiently to enable the lid to be pressed upwards to its normal position. There then remains a raw surface, which is to be covered by bringing together the edges of the V-shaped wound by means of fine pins in such a manner that the V-shaped incision becomes Y-shaped (see figs. 14 and 15).

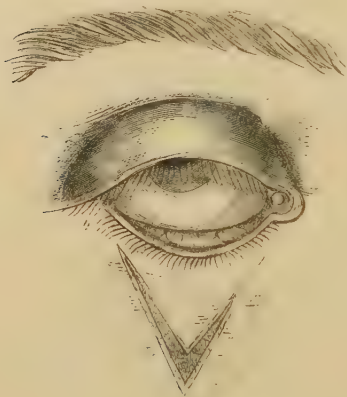


FIG. 14.—Wharton Jones's Operation
(first stage).

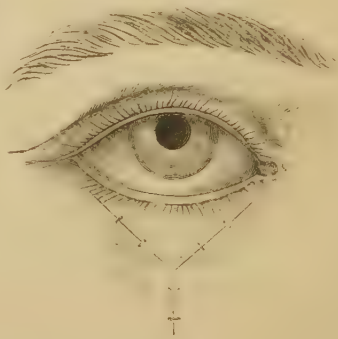


FIG. 15.—(Final stage.)

D. *Skin grafting* may be performed in various ways, either with a pedicle or without.

1. *With a pedicle*. (a) The Italian method (Tagliacozzi), in which a flap of skin is taken from the arm of the patient (or some other individual—heteroplasty) supported by an apparatus in close proximity to the eyelid and fixed to the raw surface by sutures. When it is obvious that the flap has taken root, so to speak, in its new position, the base is divided and the arm liberated.

(b) A flap from the forehead, temple, or cheek is turned so as to lie in its new bed without strangulating its pedicle or base. The raw surface from which the flap has been taken must, in its turn, receive a graft from some other part of the body where a scar will

not be noticed. Dieffenbach's operation, to be presently described, is of this nature.

2. *Without a pedicle.* (a) A large graft of skin including its whole thickness (Le Fort). It must be cut the same shape as the surface on which it is to be grafted, but of slightly larger dimensions, as it will shrink when separated from its bed.

(b) Small grafts to form separate islets of epidermal growth (Reverdin).

(c) Thin slices of epidermis and part of the cutis vera (Thiersch).

The selection of the operation must vary with the position and the state of the parts. In all cases the eyelids must be freely liberated so that they may be united by tarsorrhaphy without tension. The raw surface must then be allowed to granulate, and upon a healthy

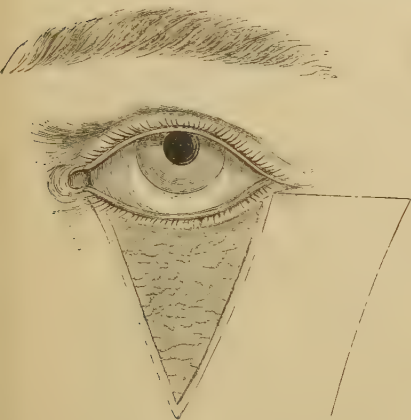


FIG. 16.—Dieffenbach's Operation for Ectropion (first stage).

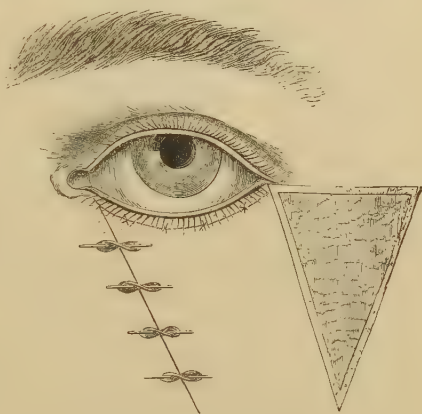


FIG. 17.—(Second stage.)

granulating surface a graft with pedicle must be chosen and implanted; if this is impossible owing to much scarring of a parchment-like character, a Thiersch's graft for choice should be applied.

In *Dieffenbach's operation* the diseased tissue is dissected away by a triangular incision, which has its base at the lower lid; a flap of skin of equal size is then marked off from the immediate neighbourhood (see fig. 16). This is loosened by careful dissection and then glided on to the recently exposed surface beneath the lower lid. It is then kept in position by fine harelip pins and sutures, as shown in fig. 17. In slight cases the surface from which the new skin has been removed soon becomes covered over by growth from

the edges of the wound ; but when a large surface is thus exposed, skin-grafts should be made from other parts of the body.

Transplantation of skin.—The operation for the transplantation of skin *en masse* in the treatment of ectropion and other deformities of the eyelids is now extensively practised. It is particularly valuable in all cases where skin is required to replace cicatricial tissue, such as that which so often follows burns of the cheek and eyelids. The operation is long and tedious ; like all blepharoplastic procedures, it requires great care and ingenuity on the part of the surgeon.

1. The mucous membrane is to be pared from the margins of both eyelids, and the raw surfaces thus produced are to be brought into apposition and united by four fine silk sutures, in order to produce temporary ankyloblepharon. The eyelashes should, if possible, be undisturbed.

2. The affected eyelid is then to be liberated from the cicatricial tissue by an incision through the skin along its whole length, parallel to its ciliary border. The contracted skin is then to be loosened by subcutaneous incision, so as to form a semilunar raw surface, or, if quite deformed and useless, it had better be dissected away. The bleeding from the surface thus exposed is to be entirely stopped.

3. A piece of skin of similar shape and about one-third larger in each diameter of the exposed surface is now to be carefully dissected from some other part of the body of the patient, or of another person. The inner side of the arm, the front of the forearm, the front of the leg, and the foreskin are all convenient parts for this dissection, which should be made as far as possible without removing subcutaneous connective tissue and fat ; great care should also be taken not to bruise the skin with the forceps used in its dissection. Before detaching the flap of skin which is to be transplanted, it is well to pass three or four sutures into different points at its margin, otherwise it shrivels up in such a manner that it cannot be opened out without difficulty and loss of time. The same sutures can be used to secure it in its new position.

The object of making the flap so much larger than the surface is that it contracts immediately after removal, as well as after union. As soon as removed it is to be transposed, and secured in its new position by numerous fine silk sutures.

4. Various methods of dressing are recommended. The plan I have adopted with success is to apply first a piece of protective, and then several layers of cyanide gauze and wool, kept in position by a bandage. The dressings should not be changed for from seven to

fourteen days. The eyelids can remain united for a longer or shorter period according to the nature of the case. When the danger of a return of ectropion is past, the eyelids can be carefully separated by incision with a sharp scalpel on a grooved director.

Congenital Deformities.—*Epicanthus*, first described by von Ammon, is a crescentic fold of skin which projects in front of the inner canthus. It is generally symmetrical, giving a broad appearance to the root of the nose. By pinching up the skin on the bridge of the nose, the epicanthus can be made to disappear. It generally improves as the child grows and the bones of the face become developed. Vision is not interfered with. Operative interference for cosmetic purposes should not be adopted during childhood. When required, it is simply necessary to remove an oval flap of skin from the median line over the root of the nose, the size of which must vary with the extent of the deformity, and to bring the edges of the wound together by sutures.

Coloboma palpebræ, or congenital cleft of the eyelid resembling harelip, is a disfigurement occasionally seen. It occurs more often in the upper eyelid, and is either partial or complete. Two may be seen in one eyelid dividing it into three folds. A dermoid growth is more often than not seen on the eyeball opposite the cleft; other congenital anomalies, as accessory auricles, harelip, coloboma of iris and choroid, microphthalmos, &c., are frequent accompaniments. If a dermoid is absent, a fold of mucous membrane like a frænum linguæ extends from the cleft to the ocular conjunctiva, reaching forward to the sclero-corneal junction. Eyelashes are absent in the cleft.

In endeavouring to explain the cause of this defect it is necessary to enter into the development of the eyelids. In a foetus of two months the eye is exposed, but towards the end of the third month a fold of epiblast immediately above and another just below the cornea develop, and eventually meet and temporarily join opposite the eye where the future palpebral fissure will be. As the fold grows forwards, its point of reflection extends backwards to form the cul-de-sac of the conjunctiva. It is believed by some authorities that the eyelid develops from two side-folds which meet and unite in

the same way as the lip is developed. Others believe that the cleft results from some adhesion having been contracted between the amnion and the eyeball, which prevents the growth of the eyelid at one or more points. The treatment is far from satisfactory. In some cases the union of the sides may be effected by paring off their mucous membrane and keeping them in apposition by stout sutures.

Dermoid cysts and *congenital ptosis* have already been described. *Ablepharon*, or absence of the eyelids, and *cryptophthalmos* of Zehender, a condition in which the eyeball is completely hidden by a fold of skin without any vestige of a proper eyelid, are very rare congenital anomalies. *Trichiasis*, *distichiasis*, *entropion*, *ankyloblepharon*, and *symblepharon* are deformities which, though more often acquired, may be congenital.

CHAPTER II.

THE LACHRYMAL APPARATUS.

ANATOMY AND PHYSIOLOGY—DACRYO-ADENITIS—TUMOURS OF LACHRYMAL GLAND—CYSTS OF GLAND—DACRYOLITHS—FISTULA OF GLAND—LACHRYMATION—EXTIRPATION OF GLAND—EPIPHORA—SLITTING UP CANALICULUS—OBSTRUCTION OF CANALICULUS—STRICTURE OF NASAL DUCT—PROBING—FISTULA OF LACHRYMAL SAC—ENCANTHIS.

ANATOMY AND PHYSIOLOGY.

THE lachrymal apparatus consists of the lachrymal gland and its excretory ducts, the lachrymal canaliculi, the lachrymal sac, and the nasal duct.

The *lachrymal gland* is placed in the upper and outer part of the orbit, a little behind its anterior margin. It consists of a large superior and a small inferior portion. The orbital portion is about 2 cm. in length, 1 cm. in breadth, and 0.5 cm. in thickness; it is lodged in a depression in the orbital plate of the frontal bone, to which it adheres by fibrous bands; its long diameter is placed transversely. The palpebral portion (gland of Rosenmüller) is much smaller and separated from the larger by a band of fascia; it is closely adherent to the back of the upper eyelid, and is covered on its ocular surface only by conjunctiva (see fig. 18).

From both portions of the gland there proceed numerous small ducts—the *lachrymal ducts*—varying from six to twelve in number; they run obliquely under the conjunctiva, and open by separate orifices into the fornix conjunctivæ at its upper and outer part.

The lachrymal gland is similar in structure to the salivary glands, consisting of acini, which contain cuboidal cells with large nuclei. In the centre of each acinus the duct begins.

The nervous mechanism of the lachrymal gland is very complex. A flow of tears may easily be excited in a reflex manner by stimuli applied to the conjunctiva, the nasal mucous membrane, the tongue, the optic nerve, &c.; and in a direct manner by the emotions.

The *lachrymal canaliculi* are two in number, situated on the margin of each lid, at the inner angle. Each commences by a small aperture, the *punctum lachrymale*, which may be seen situated on a slight eminence (papilla). Both puncta, in their natural position, dip into the depression between the eyeball and caruncle, the *lacus lachrymalis*. The upper canal is rather smaller and longer than the lower; it first ascends, and then turns downwards and inwards to the lachrymal sac. The lower canal first descends, and then runs horizontally to the sac. They usually unite and form a diverticulum (the sinus of Maier) just before reaching the sac. Near the punctum the diameter of each canaliculus is about 0.5 mm.; just beyond this

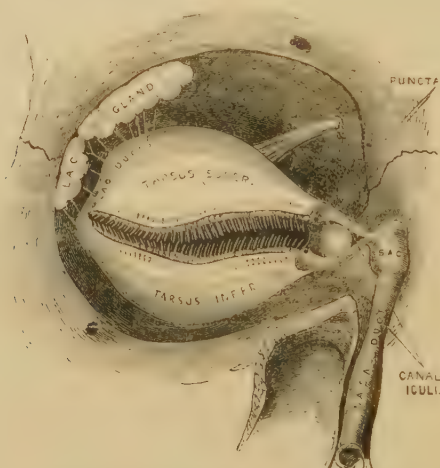


FIG. 18.—Dissection of the Lachrymal Apparatus.

it becomes suddenly dilated to 1 mm.; and for the remaining two-thirds of its course it is about 0.6 mm. Its walls are extremely thin, and are lined by stratified epithelium. Striped muscular fibres derived from the orbicularis palpebrarum encircle each canaliculus.

The *lachrymal sac* is the upper dilated portion of the passage which conveys the tears from the lachrymal canals to the cavity of the nose. It is situated in a deep groove formed by the lachrymal and superior maxillary bones. Its upper end is closed and rounded, and its lower part tapers off into the nasal duct. On its outer side and rather anteriorly it receives the canaliculi. In front of it is the tendinous head of origin of the orbicularis palpebrarum; behind, the muscular head, or tensor tarsi, and the palpebral aponeurosis.

Arching over it and running down to its inner side is the nasal branch of the ophthalmic artery. Its length is about 12 mm., its diameter 6 mm.; and at its junction with the nasal duct there is often a valvular fold of mucous membrane. It is composed of fibrous and elastic tissue, and adheres closely to the bones. It is lined by columnar epithelium, here and there showing cilia.

The *nasal duct* extends from the lachrymal sac to the inferior meatus of the nose. The osseous canal is formed by the superior maxillary, the lachrymal, and the inferior turbinated bones. This is lined by a tube of fibrous membrane, continuous with that of the lachrymal sac, and is similarly lined with columnar ciliated epithelium. At the entrance into the inferior meatus there is sometimes an imperfect valvular arrangement of the mucous membrane—valve of Hasner. The lower opening is situated 25 mm. behind the anterior nares and 25 mm. above the floor of the nose. The length of the duct varies with the development of the face; it is usually a little more than 15 mm. Its direction is downwards, and slightly outwards, and backwards.

The *lachrymal secretion* is a faintly alkaline fluid containing about 1 per cent. of solids, of which the greater part is chloride of sodium. It passes by the lachrymal ducts into the sac of the conjunctiva, where it serves to moisten the anterior part of the eye. Its exit from the sac of the conjunctiva is effected by the act of winking, which takes place at frequent intervals. In this act of closure of the lids not only is there contraction of the palpebral portion of the orbicularis, but also of those fibres which surround the lachrymal sac; thus, the palpebral fissure being closed, the tears are pressed successively through the puncta lachrymalia, the canaliculi, the lachrymal sac, and the nasal duct, into the inferior meatus of the nose, where they are evaporated by the act of respiration.

DISEASES OF THE LACHRYMAL GLAND.

Dacryo-adenitis, or inflammation of the lachrymal gland, is extremely rare. It may be acute or chronic.

Acute dacryo-adenitis, less frequently seen than the chronic, gives rise to great swelling and redness of the upper lid, especially towards the outer angle of the orbit. The swelling displaces the globe downwards and inwards, causing slight proptosis, limited movement, and, if the eyelid can be raised, diplopia. The conjunctiva is injected, and frequently there is chemosis. Pain is severe, of a throbbing nature and increased

on pressure. It may terminate by resolution, it may go on to suppuration, or it may become chronic.

The formation of abscess is indicated by increased local redness, swelling, and fluctuation.

Etiology and pathology.—As causes may be mentioned the following: injury; during the exanthemata, especially measles, and in association with mumps; as a complication of influenza, and erysipelas; also secondary to purulent and diphtheritic ophthalmia. It seems probable that in cases in which suppuration ensues the inflammation is the result of infection from the conjunctival sac. It is sometimes symmetrical, and, from its occasional association with mumps, it may be looked upon, in some instances, as a similar glandular disease, an infective dacryo-adenitis.

Treatment is simple. A free purge must be given at the onset—calomel preferred. Locally, belladonna or boric acid fomentations; and if the pain is severe, three or four leeches to the temple. Should suppuration take place, the abscess must be opened through the skin, and the cavity irrigated with an antiseptic solution. If necessary a small drainage-tube may be inserted for twenty-four hours. The wound may be slow in healing, but it is rare for a fistulous opening to remain.

Chronic dacryo-adenitis is recognised by the presence of a hard nodular growth in the neighbourhood of the lachrymal gland. It increases slowly in a forward and inward direction, *i.e.* arching over the eyeball. There is no marked redness or œdema of the eyelids. There may be no evidence of inflammation at all. The pain is slight or absent, and there is scarcely any tenderness on pressure. The eyeball is displaced, as in the acute affection, downwards, forwards, and inwards; it is limited in movement, and diplopia is often present. By involvement of the supra-orbital nerve there may be neuralgia of the scalp, or even anæsthesia. Again similar to the acute form, it may be symmetrical, first occurring in one orbit, then in the other. It usually lasts over a period of several months.

Etiology and pathology.—Chronic dacryo-adenitis has received the title of ‘mumps of the lachrymal gland’

(Hirschberg). It seems that this term would be more appropriately applied to the acute non-suppurative forms which are bilateral. The cause in many cases is obscure. Syphilis I believe to be the most common cause, and, even though a tertiary manifestation, symmetrical enlargement is the usual form met with. It may result from acquired or inherited syphilis. Primary tubercular disease of the gland has been described, but is rare; also hypertrophy of the gland, but its occurrence is extremely doubtful. Many cases of so-called dacryo-adenitis are probably inflammatory growths originating in the periosteum in the neighbourhood of the gland; and as the gland is so intimately connected with the periosteum, its subsequent involvement may be thus explained. Thickening of its capsule, or a peri-dacryo-adenitis, would constitute the bulk of the growth. Bilateral chronic dacryo-adenitis has been seen in a rheumatic subject without any other dyscrasia.

Treatment.—Our sheet-anchor in the treatment of chronic dacryo-adenitis is iodide of potassium, either alone, or in combination with mercury, whether syphilis can be ascertained as the cause or not. Locally, massage and rubbing in a stimulating ointment may be of some service.

Tumours of the lachrymal gland are also rare, and their diagnosis from the last affection is a source of great difficulty, aggravated by the fact that they also may be symmetrical. Sarcoma is the most common, and may appear as a large or small round-celled growth, as a lympho-sarcoma, adeno-sarcoma, myxo-sarcoma, chondro-sarcoma, or fibro-sarcoma. As benign growths, enchondroma, fibro-adenoma, and lymphoma have been met with. Lymphoma and lympho-sarcoma are the usual symmetrical tumours; the others are limited to one orbit. Besides being symmetrical, lympho-sarcoma infects at an early stage lymphatic glands, the pre-auricular, submaxillary, &c. The round-celled sarcomata, including lympho-sarcoma, are soft and elastic, and not likely to be easily mistaken for chronic dacryo-adenitis. The firm sarcomata—fibro-, myxo-, adeno-, chondro-, &c.—are hard, nodulated, and slow of growth, and likely to be mistaken for chronic dacryo-adenitis. The absence of any dyscrasia, of

pain, and of any sign of inflammation, assists to a very immaterial extent. The diagnosis should be made by an exploratory incision and the removal of a prominent nodule of the growth for microscopic examination.

Treatment consists in the removal of the tumour. If one of the firm sarcomata, as a chondro-sarcoma, or a benign growth, it may be 'shelled' out of its bed, as they are often encapsuled. A soft, rapidly infiltrating, round-celled sarcoma necessitates exenteration of the orbit. Success depends upon the small size of the growth and presence of encapsulation.

Cysts of the lachrymal gland are even rarer than solid growths. Instances of dermoid and hydatid cysts have been recorded, but it is doubtful if any were of intraglandular origin. *Dacryops* is the cyst of this region. It is a retention cyst, congenital or traumatic, due to obstruction in and distension of one of the lachrymal excretory ducts. A slight fulness of the eyelid is noticed, and on everting it the conjunctiva is seen to bulge as a bluish translucent tumour from the outer part of the cul-de-sac. It suddenly swells each time the patient cries, and may reach to the size of a pigeon's egg, and cause displacement of the eyeball.

Treatment.—If small, the removal of its anterior conjunctival wall will cure the condition. If large, the cyst may be removed whole by dissection through the conjunctiva; in order to obtain room the outer canthus must be divided.

Dacryoliths, or lachrymal calculi, are concretions of phosphate of lime which are apt to form in the lachrymal ducts. They are analogous to the concretions ('gouty deposits') sometimes seen in the Meibomian ducts. On everting the eyelid they become apparent, and are easily liberated by an incision through the conjunctiva immediately over them.

Fistula of the lachrymal gland is either congenital or the result of injury or of abscess. A small opening exists in the skin near the upper and outer angle of the orbit, through which the tears almost constantly escape. The flow of tears through this abnormal passage may be increased by irritation of the conjunctiva.

Treatment.—The edges of the opening will sometimes unite by the application of solid nitrate of silver every few days, or

by the introduction of a wire of the thermal cautery at a dull red heat. These measures may be assisted by previously establishing an artificial opening into the sac of the conjunctiva by the introduction of a seton in the region of the lachrymal ducts. Some cases are obstinate, and require ultimate extirpation of the gland.

Lachrymation, or the overflow of tears from excessive secretion, must not be confounded with *epiphora*, a term almost exclusively used to denote overflow from obstruction or some interference with the passages of exit. Excessive secretion may be physiological, *e.g.* emotional. Hysteria, and even some organic diseases of the brain, may be attended with profuse lachrymation. It is, however, more often the result of reflex irritation, as foreign bodies, or abrasions, in the conjunctiva or cornea, nasal irritation, pungent tastes, &c. The different forms of ophthalmia and most cases of keratitis will cause excessive lachrymation. Internal ocular troubles, as iritis, choroiditis, retinitis, do so to a very slight extent, sometimes not at all. Instances of bloodstained tears have been observed in which hysteria and injury, self-inflicted or not, could be excluded.

Operation for Extirpation of the Lachrymal Gland.—The brow shaved off and the part cleansed, the patient is to be anæsthetised. Instruments required are a small scalpel, a horn spatula, a vulsellum forceps, a director, toothed forceps, and artery forceps. The operator should stand to the patient's right, his assistant to the left. Strict attention to antisepsis must be paid throughout. An incision is made parallel to, and through the lower part of, the eyebrow, extending from the middle of the upper edge of the orbit to its outer angle. Skin and orbicularis are divided and the periosteum exposed. Below the orbital rim the palpebral aponeurosis will be seen, and must be carefully divided close to the edge of the orbit and secured with a suture. The lachrymal gland, if large, will now present itself to view; if small, it will be found deeply seated in the lachrymal fossa. In either case it must be firmly seized with the vulsellum forceps and separated from its surroundings with a director. The dissection should be begun from the orbital surface of the gland. In clearing it from its ocular relations, great care should be taken to *avoid laceration of the levator palpebræ muscle*. The palpebral aponeurosis must be sutured, and then the edges of the wound are to be

brought together by fine silk or catgut sutures, and a light antiseptic dressing applied.

DISEASES OF THE EXCRETORY PASSAGES.

Affections of the drainage system may be classified as those of the puncta lachrymalia, those of the canaliculi, and those of the lachrymal sac and nasal duct. In each of these affections *epiphora*, or overflow of tears, is a troublesome symptom, which is always aggravated by exposure of the eye to cold or wind, or by any cause which would increase the secretion of the lachrymal gland.

Epiphora is a symptom of the following disorders: stenosis, or absence, of the puncta lachrymalia, eversion of the lower punctum from facial palsy or senile ectropion, displacement of the puncta from cicatrices, obstruction of the canaliculi by foreign bodies—as for example, eyelashes, chalky concretions, warty growths, lepto-thrix buccalis, &c.—or they may be obliterated by the contraction of scars, or from pressure by growths or inflammatory swellings, as inflammation of the caruncle (en-canthis), chancre, sarcoma, gumma, rodent ulcer, lupus, &c., and, finally, any obstruction in the lachrymal sac or nasal duct.

The punctum may be congenitally small, or absent; or stenosed or obliterated from chronic blepharitis. It may be everted in elderly subjects from laxity of the lower lid combined with chronic thickening of the conjunctiva. In facial paralysis the lower lid may be, from want of muscular tone, away from the eyeball.

Treatment.—Stenosis of the punctum is cured by dilatation. It may have to be repeated. If both puncta are absent an entry may be made into the sac with a Graefe's cataract knife introduced into the sac immediately behind the tendo oculi, and nearer the lower lid. If, in elderly subjects, the punctum cannot be restored to its proper position, the best method of restoring conduction of the tears into the lachrymal sac is by slitting up the lower canaliculus, so that they may enter its channel nearer the sac.

Operation for Slitting up the Lower Canaliculus.—A general anæsthetic is not required, except in the case of children and persons

of nervous temperament. In all cases, however, it is well to inject a 4 per cent. solution of cocaine hydrochlorate into the lachrymal sac by means of a lachrymal syringe about five minutes before operating. Patient to be seated on an ordinary chair with the head thrown back, so that the face looks towards the ceiling. Operator to stand behind the chair. The lower lid is to be drawn taut, and slightly everted by the thumb of one hand (fig. 21), while with the other hand the probe point of a canaliculus knife (fig. 19) is introduced vertically. When the knife has well entered the canaliculus, its point is to be directed inwards, and slightly backwards in the direction of the lachrymal sac, until it reaches the inner wall. The edge of the knife during its passage is to be turned upwards and inwards, so as to divide the canaliculus close to the muco-cutaneous junction. When the knife has thus reached the inner wall of the sac, it must be boldly brought up from the horizontal to the vertical position, the eyelid being still kept taut by the opposite thumb. Thus the whole length of the canaliculus is divided quite into the sac.

Another method of slitting up the canaliculus may be adopted. A fine grooved director (Critchett's) is passed through the canaliculus, along which a fine knife is passed into the sac.

The *upper canaliculus* may be divided, if necessary, in a similar manner. It is rather more difficult to enter the upper punctum. Weber, Arlt, Story, and others prefer to open the upper canaliculus in preference to the lower, for the following reasons: (1) probes can be passed more easily; (2) the loss of the punctum is less felt in the upper, as the greater volume of tears pass along the lower canaliculus. The benefits derived appear to me more imaginary than real. If the stricture is cured, there is no greater danger of persistent epiphora in operating on the lower canaliculus than the upper, nor is the passage of probes in competent hands more difficult. The division of the upper canaliculus, on the other hand, is certainly more difficult than that of the lower. Here the upper lid must be made tense as the probe-pointed knife is passed downwards and inwards to the sac.



FIG. 19.
Weber's Canaliculus Knife.

Various modifications of the knife represented in fig. 19, such as Bowman's, Wecker's, Liebreich's, and others, are used. It sometimes happens that the punctum is very small, and will not admit the probe-point of the knife; in these cases a fine conical probe should be first introduced, by which means it may be sufficiently dilated to admit the knife.

Obstruction of the lower canaliculus is not unfrequent. It may be caused by inflammation of the mucous membrane extending from the conjunctiva, or by the presence of a foreign body such as an eyelash. It is sometimes due to chalky concretions or collections of fungi, and is often the result of cicatrix following burns, and lacerated wounds. Warty growths of very small size, but sufficient to cause troublesome epiphora, may be found in one or both canaliculi. Abscesses and tumours may, by their pressure, occlude the passage. The upper canaliculus is less frequently affected except in the case of wounds and burns.

Treatment.—This must vary as the cause of the obstruction or obliteration. An eyelash usually projects a short distance out, surrounded with a coating of mucus, and may readily be removed with epilation forceps. It produces a characteristic inflammatory patch on the ocular conjunctiva from irritation. A chalky concretion, visible through the translucent wall, may be removed by a small incision, without danger of traumatic stricture if the incision is made parallel to the canaliculus. If nothing can be seen, the nozzle of a lachrymal syringe should be introduced and water injected. If it regurgitates through the opposite canaliculus the obstruction is in the duct; but if it returns by the side of the nozzle without entering the sac, the obstruction is in the canaliculus. It may be felt in using the lachrymal syringe; if not, the punctum should be dilated, and the dilator gradually forced in the direction of the sac. A very frequent point of obstruction is just at the entrance to the lachrymal sac. This is indicated by the movement of the whole lower lid when the knife or probe is pressed in that direction. Firm pressure will generally overcome this resistance.

Stricture of the nasal duct is the most common affection of the lachrymal apparatus.

Cause.—The original cause of this affection is frequently difficult to make out. It appears in many cases, however, to commence by extension of catarrhal inflammation of the lining membrane of the nose. It is possible for it to be caused by extension from the conjunctiva through the lachrymal sac, but more often it is the obstruction which causes the conjunctivitis. In strumous and syphilitic subjects, periostitis of the bones forming the nasal canal is a frequent cause of obstruction by extension of inflammation to the muco-periosteal lining. It may also be caused by injury to the nasal bones, and by the existence of carious teeth in the upper jaw. Pressure, causing more or less obliteration of the canal, by tumours of various kinds, as fibro-sarcoma, myxoma, and exostosis developed in the upper jaw, the antrum, or the nasal fossa, is not uncommon.

The seat of the stricture is usually at the upper part of the tube just below its junction with the lachrymal sac, but it may be situated anywhere in its course.

The *symptoms* of stricture of the nasal duct are very variable. In some cases the only observable departure from the normal condition is an overflow of the tears, which is increased by exposure to cold wind or bright light. There may be little or no inflammatory redness of the conjunctiva. The puncta lachrymalia and canaliculi are found to be quite patent, and in their normal position. There is no perceptible tumour in the region of the lachrymal sac. On making firm pressure with the finger over the region of the sac, there may be no regurgitation of its contents; more frequently, however, there is some reflux of a viscid secretion through the canaliculi, which may be quite clear and colourless, or may be more or less purulent, but is always of a more tenacious character than the tears. In the majority of cases there is distinct *swelling of the lachrymal sac* (chronic dacryo-cystitis, mucocoele). The amount of swelling varies from a mere fulness to an absolute protuberance of the skin just below the internal palpebral ligament. Firm pressure with the finger over this will usually cause the dispersion of its contents either upwards through the canaliculi, or downwards through the nasal duct. The nature of this liquid varies according to the gravity of the lesions of the sac; it may be simple mucus, or muco-pus, or even pus. Epiphora

is troublesome, causing irritation and redness of the conjunctiva and eyelids. The swelling is usually free from pain and devoid of tenderness, even on pressure; it is localised, and increases but slowly. It is, however, liable at any time to take on an active state of inflammation and suppuration, thus constituting *abscess of the lachrymal sac* (acute dacryo-cystitis), in which the symptoms are altogether more severe. The swelling now becomes suddenly increased, and of a tense brawny nature. The root of the nose, the lower part of the frontal region, the upper part of the cheek, are red and œdematous; the eyelids, also, are much swollen. The skin over the region of the lachrymal sac and the surrounding parts is of a dusky red colour. There are intense local pain and heat, in addition to the redness and swelling. General symptoms, such as pyrexia, rigors, and vomiting, may occur. This kind of inflammation of the sac never terminates in resolution; suppuration first takes place inside the sac, forming an abscess, which soon perforates its walls, setting up inflammation of the surrounding cellular tissue.

Perforation of the wall of the sac is attended by diminution of the pain, which, although it does not disappear, becomes greatly lessened in intensity. Then commences the more serious inflammation and swelling of the tissues around the sac and in its vicinity, leading to the formation of abscess, which, if untreated by surgical interference, usually terminates by pointing through the skin about 1 cm. below the lower punctum lachrymale. This opening gives exit at first to purulent matter, which gradually decreases as the inflammation and swelling subside. It may heal up of its own accord, but generally remains as a *fistula of the lachrymal sac*, giving exit at first to the purulent matter, then to muco-pus mixed with the tears, and finally to the tears alone, which ought to have passed down the nasal duct.

The *diagnosis* of swelling of the lachrymal sac is easily made when there is but little inflammatory trouble. Its situation, its history and accompanying lachrymation, its more or less complete disappearance on firm pressure, serve to distinguish it from other tumours of this region. When inflammation is severe, it may at first simulate erysipelas of the eyelids, but in

abscess of the sac we have seen that the redness is most intense over the seat of inflammation, and shades off and becomes simple œdema of the surrounding parts, that there is always a history of epiphora, and generally of tumour of the sac. In erysipelas, the redness is equal all over the swelling, its outer edge is seen to spread to surrounding parts, and there is no history of previous lachrymation or tumour. It may be difficult to distinguish it from a chalazion occurring near the inner canthus in the upper lid. In any case, the canaliculus should be syringed, when it will be at once evident whether any obstruction exists or no. It may also be difficult to say whether an abscess at the inner angle of the eye had its commencement within or outside the sac. Here, again, the previous history of overflow of tears and the presence of a tumour of the sac are useful aids to diagnosis, and all doubt can often be dispelled by pressure over the swelling, when a regurgitation takes place through the puncta lachrymalia.

Stricture of the nasal duct also may give rise to the development of serious lesions of the cornea, conjunctiva, and eyelids. After prolonged obstruction, a chronic inflammation of the conjunctiva is often established. This may spread to the edges of the eyelids, causing blepharitis and even ulceration. The cornea often becomes affected with superficial, ill-defined, greyish-white opacities and ulcers. Any operation involving wound of the cornea, such as that of iridectomy or extraction of cataract, that might be performed under this condition of stagnation in the sac, would be seriously interfered with; the wound would become infected by the organisms in the sac, and suppurative panophthalmitis easily provoked.

Treatment must be directed to the permanent cure of the stricture.

I. *When there is no abscess of the sac, but only swelling, or even only epiphora*, the lower canaliculus should be slit up in the manner indicated above (p. 50), and a probe should be passed through the stricture at once. It should be passed again within forty-eight hours to prevent the closing up of the canaliculus, and the operation should be repeated twice or thrice a week until the epiphora has ceased, and all symptoms

of obstruction have disappeared. Even then it is well to continue the probing once a week for a few months.

The kind of probe used is a matter of little importance so long as it is of the right calibre, and is passed in the proper direction—viz. downwards and rather outwards and backwards. Many varieties of probes are now in use. The bulbous probes

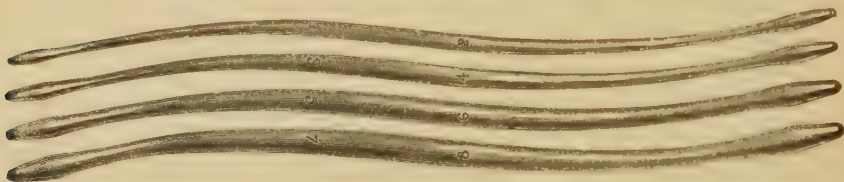


FIG. 20.—Set of Probes for the Nasal Duct.

of Bowman are curved in opposite directions towards each end, and instead of being of equal calibre throughout they are bulbous towards each extremity, as shown in fig. 20. These probes number from 1 to 8, No. 1 being about 1 mm. across the bulb, No. 8 about 3·5 mm., and the remainder of intermediate sizes. There are numerous other varieties of probes which it is not necessary to describe.

The mode of introducing the probe is similar to that of introducing the canaliculus knife (see fig. 21). It is passed horizontally along the canaliculus until it reaches the inner wall of the lachrymal sac, the lower lid being kept tense by the thumb of the opposite hand. The probe is known to be well inside the sac by the resistance offered by the lachrymal bone, and by the absence of dragging on the skin of the lower lid. The end of the probe being kept in contact with the inner wall of the sac, it must now be brought from the horizontal to the vertical position and pushed down the duct. The direction of the duct, as we have seen, is downwards and slightly backwards and outwards; fairly firm pressure can be made in this direction so long as the probe is kept in contact with the inner end of the brow. I usually commence with a probe of 2 mm. diameter; if this passes easily, I try the next size larger; if it does not pass without great force, I try smaller sizes until one is found which will pass through the stricture. It seldom happens that a stricture is so tight that it will not admit a probe of 0·5 mm. diameter. Thus we are enabled to form an estimate of the extent and the nature of the constriction. One of three methods can now be adopted—viz.:

1. *Gradual dilatation*, by slightly increasing the diameter of the probe used at each sitting. 2. *Rapid dilatation*, by the passage at one sitting of No. 1 probe to No. 6, and continuing this practice at after-sittings. 3. *The incision of the stricture by means of a knife*,

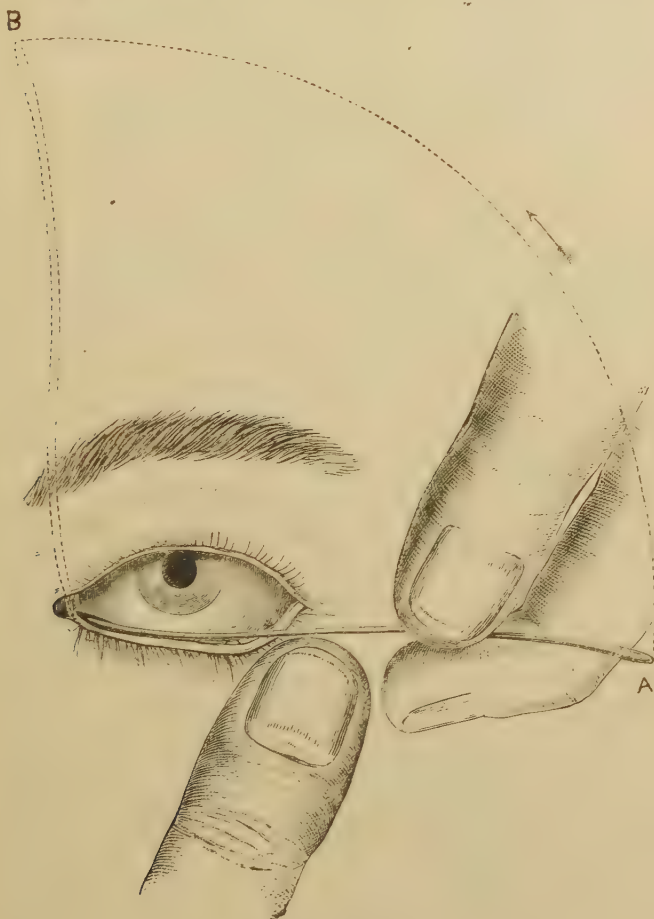


FIG. 21.—Probe in First and Second Positions.

and the subsequent passage of probes. This is of great service in very tight strictures. The best knife for this purpose is that of *Stilling* (fig. 22). It is introduced into the lachrymal sac in the same way as the probe, and then forced down in the direction of the duct two or three times in succession, the blade being turned in different directions

at each passage, after which probes of 1 mm., 2 mm., or 3 mm. can be passed. Other knives, such as those of Bowman and Weber, can be used for this purpose, but, owing to their brittleness and delicacy, their blades are apt to be left in the stricture.

II. *When there is abscess of the sac*, and fistula has not yet formed, an immediate effort should be made to give free exit to the



FIG. 22.—Stilling's Canaliculus Knife.

pus. This should, if possible, be effected by slitting up one of the canaliculi ; if, however, the swelling is so great as to prevent this, a puncture should be made by thrusting a small scalpel through the skin 1 cm. below the inner canthus, the direction of the cut being downwards and outwards. When fistula has been established by rupture of the abscess, or when the abscess has been opened by incision and the swelling has subsided, the lower canaliculus should be slit up so as to establish a free exit for any pus that may yet be retained in the sac or may be afterwards formed. Probing must now be attempted ; but should there be any difficulty of introduction to the nasal duct, it is well to wait a few days for subsidence of inflammatory swelling of the mucous membrane of the sac and duct. Then a small probe can generally be introduced, and gradual or rapid dilatation or incision by Stilling's method may be performed. These inflammations of the sac, whether chronic or acute, very frequently yield to treatment by dilatation only ; but in some cases, especially where there has been much suppuration, the cure is often

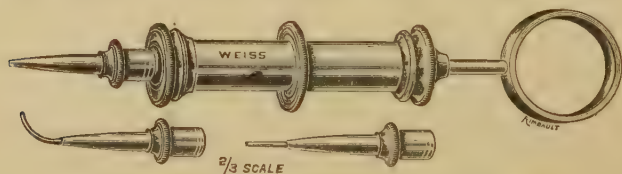


FIG. 23.—Anel's Syringe for injecting Lachrymal Sac.

accelerated by local astringents. An excellent astringent and antiseptic for this purpose is to be found in the use of a 2 per cent. to 4 per cent. solution of pure boracic acid. It should be injected into the sac by means of a syringe, the nozzle of which (fig. 23) can be easily introduced. Other solutions than that of boracic acid

can be used for this purpose, such as those of alum, sulphate of zinc, and lapis divinus of the same strength. Solution of nitrate of silver of strength $\frac{1}{2}$ per cent. is beneficial in some cases, more especially where the discharge is markedly purulent.

In children, nervous adults, and where there is a tendency to closure by cicatrisation at the entrance to the sac, the insertion of a small silver or lead style of the shape shown in fig. 24 is very convenient. A probe should first be passed to ascertain the length of the duct, and a style of proper length being chosen, its upper end should be bent at right angles to the extent of 4 mm. or 5 mm. It is then introduced so that its lower end rests on the floor of the nose, and its upper bent portion lies in the groove of the open canaliculus. After



FIG. 24.—Style for Nasal Duct.

its introduction it must be watched lest the parts become inflamed, in which case it must be removed and re-inserted after a few days; if the parts remain quiet, it can be kept *in situ* for several weeks, and will be found to be of great service, the epiphora being often improved even whilst the patient is wearing the style. This method is also useful in ordinary cases where the passage of the probe is inconvenient or impossible owing to the patient living at a distance, or being unable to attend. Styles are often made of lead wire and moulded so as to fit the individual case. If it proves comfortable and does not show, a gold one may be made according to the lead pattern and retained in the duct for several months. T. H. Bickerton has introduced a combined hollow gilt style and probe. The probe can be passed first, and the style slipped over the upper end and passed down into position. The probe may be given to the patient to pass down through the style once a week, or more often if required.

In certain obstinate cases, where overflow of the tears still persists after all the efforts above indicated have failed, the extirpation of the lachrymal gland is recommended. The removal of this organ (see p. 49) has been repeatedly performed without injurious results; and the operation is well spoken of by Lawrence, Abadie, and other surgeons.

Obliteration of the lachrymal sac, by excision, by the actual cautery, or strong caustics, as the potassa cum calce, chloride of zinc, &c., is also occasionally practised by some surgeons in obstinate cases.

Excision is not an easy operation, and necessitates division of the tendo oculi. Before caustics are applied the sac must be laid freely open from the front, and care must be exercised to limit the

action of the corrosive so that it does not damage the eye or cause necrosis. I prefer to open the sac from the front, and with a sharp curette scrape away the whole lining membrane. The fundus, or that part above the tendo oculi, is liable to be left behind as it extends much higher than in the normal condition, in which case the operation will probably have to be repeated.

Fistula of the lachrymal sac may be congenital, but is much more frequently the result of neglected inflammation. It consists of a sinus extending from the sac to the skin just below (about 1 cm.) the inner canthus. The opening is usually small and gives passage to the tears and mucus which ought to pass down the nasal duct. The skin and subcutaneous tissue in the vicinity of the fistula may be little affected, but is usually swollen and red; sometimes there is chronic ulceration, with much induration of the tissues surrounding it.

Treatment must first be directed to the stricture (p. 55). This being so improved that the tears can flow through the nasal duct, the fistula may heal without further treatment. If ulceration exists, the coarse sprouting granulations should be scraped away with a curette, the dusky overlapping skin trimmed off with care, and healthy granulations promoted. If the area is large, skin grafts should be utilised to prevent much cicatricial contraction (see p. 38).

Encanthis.—The plica semilunaris and caruncula lachrymalis may be the primary seat of disease. They are, as parts of the conjunctiva, invariably involved to a greater or less extent in acute inflammatory affections of that membrane. Sarcomata and various benign growths have originated in the caruncle. The term *encanthis* has been used to embrace all diseases of this body. Supernumerary caruncles are occasionally seen.

CHAPTER III.

THE CONJUNCTIVA.

ANATOMY AND PHYSIOLOGY—HYPERÆMIA—CATARRHAL CONJUNCTIVITIS—
 PURULENT CONJUNCTIVITIS—GRANULAR CONJUNCTIVITIS—MEMBRANOUS
 CONJUNCTIVITIS—PHLYCTENULAR CONJUNCTIVITIS—SPRING CATARRH—
 OPHTHALMIA NODOSA—TUBERCULOSIS OF THE CONJUNCTIVA—SYPHILITIC
 AFFECTIONS—PTERYGIUM—PINGUECULA—AMYLOID DEGENERATION—
 XEROSIS—PEMPHIGUS—TUMOURS.

ANATOMY AND PHYSIOLOGY.

THE healthy conjunctiva varies very slightly in different individuals; in the dark races it is the rule to find it somewhat pigmented, in white people it is the exception.

For convenience of description the conjunctiva is divided into three portions—the *palpebral*, the *ocular*, and the *culs-de-sac*; the latter are the reflections of the membrane.

The *palpebral* conjunctiva lines the posterior surface of each lid, and has already been described (p. 5). Its vascularity, apart from local causes, affords a valuable indication of the state of the circulatory system, being blanched in anæmic subjects, bright red in plethoric persons. The *ocular* conjunctiva covers the anterior third of the globe, and is subdivided into *scleral* and *corneal*.

The scleral portion is a thin, loose, and almost transparent membrane which glides freely over the subjacent sclera and capsule of Tenon. At the sclero-corneal junction it is slightly thickened (*limbus conjunctivæ*), and in it the conjunctival vessels terminate in vascular loops, which, under pathological conditions, bud out between the corneal epithelium and Bowman's membrane, giving rise to the condition known as *pannus*. Œdema of the limbus causes it to swell and overlap the cornea (*chemosis*). Above and below, the ocular conjunctiva is continuous with the fornices. Internally, it forms a semilunar fold (*plica semilunaris*), upon which, and slightly internal to it, is situated a small granular and vascular elevation (*caruncula lachrymalis*); these constitute Nature's steps for the exit of foreign bodies, which, having entered the palpebral sac, are,

by the action of the orbicular muscle, and by the flow of tears, directed towards the inner canthus. The plica semilunaris is considered to be the rudiment of the *membrana nictitans* or *palpebra tertia*, present in birds and most quadrupeds. The concavity of the fold points outwards. The caruncle is composed of vascular connective tissue covered by modified skin, upon which a few fine hairs may be seen. The conjunctival vessels are derived chiefly from the muscular and lachrymal branches of the ophthalmic artery. They can rarely in a healthy membrane be traced to the corneal margin; in inflammatory changes, however, the extreme vascularity of the ocular conjunctiva becomes apparent. The microscopic structure consists of stratified epithelium supported upon a loose fibro-vascular membrane, in which nerves and lymphatic vessels are found. The latter form a well-developed network, diminishing in size towards the corneal margin, where they are connected with the cell-spaces of the cornea. The corneal conjunctiva is entirely epithelial (see Cornea).

The *fornices* (culs-de-sac, retrotarsal folds) of the conjunctiva are the reflections of that membrane from the eyelids on to the globe. The upper fornix is a favourite hiding-place for foreign bodies, collections of mucus, pus, and the growth of micro-organisms. It is an interesting fact that bacteriologists are of opinion that no pathogenic organism is present in a healthy conjunctival sac. The pathogenic properties of the *staphylococcus epidermidis albus*, a constant inhabitant of this sac, are practically *nil*. Their value lies in shutting off the cavity of the orbit from the external world, and, at the same time, from their laxity, offering no impediment to the movements of the eyeball. The lower cul-de-sac is readily exposed by drawing down the lid when the patient is looking up. This is not the case with the upper, which is hard to examine thoroughly; after everting the upper lid, gentle but firm pressure is made upon the eyeball in an upward and backward direction, the patient looking down all the while, and the cul-de-sac will prolapse. The pressure on the globe, of course, is made with the intervention of the lower lid. This method is, after a little practice, readily acquired. The membrane in the culs-de-sac is thicker than elsewhere, and is arranged in transverse rugæ.

Microscopically, this portion of the conjunctiva consists of epithelium and subepithelial tissue; the former is of the stratified variety, but the arrangement of its cells differs from the epithelium on the palpebral and ocular conjunctiva. The deepest cells are short and cuboidal, the surface cells are triangular with a long apical process extending downwards towards the limiting membrane; intervening are spindle and polygonal cells. The subepithelial tissue is com-

posed mainly of fibro-vascular connective tissue, containing a few elastic fibres, and here and there a lymphoid follicle. Lymphoid follicles can, in a healthy membrane, always be found in the lower cul-de-sac, whereas very few, if any, exist in the upper. The sub-epithelial tissue of the upper fornix is limited by the muscle of Müller, some fibres of which are reflected around it into the ocular conjunctiva; similar unstriped muscle-fibres limit the deeper strata of the lower cul-de-sac.

HYPERÆMIA OF THE CONJUNCTIVA.

Hyperæmia of the conjunctiva is very common, and is to be distinguished from inflammation by the absence of discharge. There is often a slight collection of mucus at the inner canthus each morning, but never any muco-purulent secretion. The palpebral conjunctiva is redder than natural, and there is often a slight local redness of the ocular conjunctiva to the outer side of the cornea. The symptoms exceed the signs. A sensation of dust or sand in the eyes, smarting, lachrymation, the eyes feeling hot, inability to read for any length of time, and a weightiness of the eyelids, are a few of the manifold symptoms complained of and yet scarcely to be explained by the slight vascular disturbance seen. The causes are many and varied. Age, sex, occupation, the weather, the state of the general health, the presence of secondary syphilis, are a few points to be considered. If the portal system is deranged, there will be hyperæmia of the conjunctiva. Alcohol, excessive smoking, the glare of the fire, and failure of accommodation either from refractive errors or old age; the use of certain drugs, as the iodides, the bromides, arsenic, &c.; irritation from foreign bodies and pungent vapours, are some of the many causes. *Passive congestion* is seen in cases of orbital tumours, thrombosis of the cavernous sinus, glaucoma, and in certain diseases of the heart and respiratory system.

Treatment.—The discovery and removal of the cause, the use of some bland and cooling lotion as boric acid, local rest—*i.e.* care not to strain the eyes—and proper night rest.

INFLAMMATION OF THE CONJUNCTIVA.

Inflammation of the conjunctiva (*ophthalmia, conjunctivitis*) presents a variety of more or less typical forms, although

cases often occur which seem to occupy the borderland between one form and another. The chief classes are :

1. Catarrhal conjunctivitis { *a.* Simple.
 b. Follicular.
2. Purulent conjunctivitis { *a.* Gonorrhœal ophthalmia.
 b. Ophthalmia neonatorum.
3. Granular conjunctivitis.
4. Membranous conjunctivitis { *a.* Croupous.
 b. Diphtheritic.
5. Phlyctenular conjunctivitis.
6. Spring catarrh.

1. Catarrhal Conjunctivitis.—(*a*) *Simple catarrhal conjunctivitis* may be either chronic or acute. The former, in many instances, is hardly to be distinguished from congestion of the conjunctiva. The latter is an acute contagious disease.

(1) *Chronic catarrhal conjunctivitis* is of very frequent occurrence, and presents itself under different aspects.

Symptoms.—In mild cases the symptoms are not well marked : there is slight redness of the palpebral conjunctiva and of the fornices, increased secretion of mucus, sticking together of the lids on awaking in the morning, and a sense of discomfort and gritty feeling in the eyes, more especially in the evening and by artificial light. In more severe cases these symptoms are exaggerated ; the patient complains of difficulty in keeping the eyes open, especially in the evening, although they may feel fairly well by day. There is a sense of pricking and irritation in the eyes as if from the presence of an eyelash or of sand in the eyes. The vision, also, is troubled, and artificial lights are often surrounded by haloes or rainbow colours. Other unpleasant sensations may occur, as burning, itching, early fatigue in using the eyes for fine work. In the morning the eyelids are gummed together by a dry yellowish secretion, which can be seen at the roots of the lashes and at the inner canthus. It chiefly occurs in adolescents and in old people. Its duration is variable, but frequently very persistent and difficult of cure.

Complications may occur if proper treatment is neglected ; the chief of these is inflammation of the edges of the lids

—blepharitis—owing to the irritation caused by increased lachrymation, and by adhesion and incrustation. The overflow of tears also causes eczematous contraction of the lower lid, so that it becomes everted and the inferior lachrymal punctum displaced.

Causes.—(1) A previous attack of acute catarrhal conjunctivitis which has passed into a chronic stage. (2) Defective hygienic conditions, vitiated air, smoke, dust, crowded dwellings, sitting up late at night, excessive indulgence in strong drink. (3) Overwork of the eyes, especially by hypermetropic and astigmatic subjects, undoubtedly leads to hyperæmia of the conjunctiva, and so tends to produce chronic catarrh. (4) Local irritation, as from the presence of a foreign body in the palpebral sac. (5) A gouty constitution, especially when associated with an overloaded portal system and deficient exercise. (6) The lachrymal passages are often wanting in patency.

Treatment consists : (1) In the removal as far as possible of all local and general causes of irritation. Proper regulation of the mode of life and of hygienic conditions. (2) In mild cases, the application of a 1 per cent solution of nitrate of silver to the everted lids at intervals of three or four days, combined with the use of cold wet compresses of 2 per cent. solution of boric acid applied for fifteen minutes three times daily, and the use of simple ointment to prevent adhesion of the lids during sleep. In the more severe cases the same treatment applies, but the silver solution should be stronger (2 per cent.) and the lotion of a more astringent nature, such as sulphate of zinc, lapis divinus, tannin, alum, and others—from $\frac{1}{6}$ to 1 per cent. It is well to change these lotions from week to week, as they seem to lose their beneficial effect when used for a long period. The wet lint should be applied loosely over the eyelids, and the patient should open the eyes so as to allow the lotion to enter the palpebral sac. (3) It is always well, especially where epiphora is complained of, to syringe the lachrymal sac and nasal duct with the lachrymal syringe (fig. 23) in order to be sure that there is no obstruction.

(2) *Acute catarrhal conjunctivitis* (catarrhal ophthalmia, muco-purulent conjunctivitis, school ophthalmia, blight).

Symptoms.—The conjunctiva of the lids is intensely

injected, and in more severe cases the ocular conjunctiva is also affected. According to the degree of severity of the attack, so is there much or little swelling of these parts as well as of the plica semilunaris and of the eyelids. Frequently small ecchymoses can be seen on the swollen mucous membrane. There are increased secretion of tears, and a copious mucopurulent discharge. The lymph appears in small or large flakes, floating in the lower cul-de-sac. The more severe the inflammation, the greater is the amount of discharge and the more purulent its character; indeed, in the severe forms of acute catarrhal conjunctivitis, where there are much purulent discharge and great œdema of the lids, it is sometimes difficult, at first, to distinguish the affection from gonorrhœal ophthalmia. During the course of a few days, however, the swelling subsides, the discharge diminishes, and the diagnosis is assured. Any doubt can be immediately laid aside by the bacteriological examination of the discharge. The lids become firmly closed during sleep, so that they can only be opened after fomentation with warm water. The patient complains of copious lachrymation, intolerance of light, pricking and burning in the eyes. The intensity of these varies with the intensity of the attack. As a rule, there is not much pain, unless some complication, such as ulcer of the cornea or iritis, occurs. The sensation, generally, is that of a foreign body in the eye. The vision is much disturbed by the presence of mucus or mucopus upon the cornea. All the symptoms are more marked in the evening than in the morning, especially by artificial light.

This affection is exceedingly contagious, and often spreads through a household, or becomes an epidemic. It usually attacks both eyes, either simultaneously or within a few days of each other, and, after a period varying from eight to fourteen days, yields to treatment, or even disappears spontaneously. Complications are rare, but phlyctenules or corneal ulcers may develop. Small grey ulcers appear at the edge of the cornea, which in a few days may unite so as to form a crescentic ulcer involving a considerable portion of the circumference.

Etiology and pathology.—It is now universally believed that acute catarrhal ophthalmia is caused by a specific organism. It is a most contagious affection, has a definite incubation

period of twenty-four hours, runs a definite course, and terminates, in most instances, in complete recovery. A small bacillus has been described by Weeks, Kartulis, Morax, Stephenson, and others. The organism of these investigators appears to be identical. A pure cultivation has reproduced the ophthalmia in the human subject. I have myself on several occasions, in conjunction with John Griffith, found a similar organism in the discharge taken from typical cases. It is a very small bacillus, smaller than the xerosis bacillus, readily stained with methylene blue (see p. 72), does not respond to Gram's stain, occurs in clusters in the cells and in the intercellular fluid, and frequently exhibits a slight central constriction. A larger organism has been recently described by Gelpke,¹ which he calls '*bacillus septatus*' and found present in two epidemics of ophthalmia. There is every reason to believe that Gelpke's organism is identical with the harmless xerosis bacillus.

Another organism, however, which has been proved to be in direct causal relationship to the disease, as shown by Gifford² and substantiated by others, is the pneumococcus of Fraenkel. Even the bacillus diphtheriæ has been found in simple catarrhal ophthalmia.

It is probable that the disease is spread only by contagion, though it is still upheld by some that the infection may take place through the medium of the atmosphere. Outbreaks of ophthalmia are more common in springtime and autumn, and in the latter more especially after a dry summer. This was very evident in the autumn of 1892. After an exceptionally dry summer several outbreaks came under my immediate notice. A fact of considerable importance is the belief of some surgeons, notably Hutchinson, that school ophthalmia is acute trachoma, a mild expression of the disease, but liable to develop into the serious type. If this be true it would be a gross error of judgment to regard it lightly; but, in opposition to this view, it is possible to completely stamp out an outbreak without a single case becoming chronic, and without a recurrence. This, apart from the absence of the local signs, is unlike the clinical picture of trachoma.

¹ Gelpke, Graefe's *Archiv*, xlii. 4, p. 97.

² Gifford, *Archives of Ophthalmology*, 1896.

Treatment.—The best and surest remedy is to brush the everted lids and culs-de-sac with a 2 per cent. solution of nitrate of silver. The mucous membrane should be gently dried with a piece of clean lint before applying the silver. This immediately produces a thin layer of albuminate of silver over the surface touched. The excess should be washed away with water before the lids are replaced. The brushing should be repeated every morning. It is followed by considerable pain and irritation after the application, but these usually pass away in the course of half an hour, and the patient experiences relief, whilst the mucous membrane looks paler and less inflamed. When the patient cannot be seen daily by the surgeon, he may be allowed to use a weaker solution of nitrate of silver ($\frac{1}{4}$ to $\frac{1}{2}$ per cent.) in the form of drops. This is less satisfactory, as the drops must come in contact with the cornea, and the weak solution is slower in action.

In addition to the nitrate of silver, it is well to place cold compresses of a solution of boric acid (2 per cent.) upon the eyelids for half an hour, every few hours, and to use a simple ointment to the edges of the eyelids to prevent their adhesion during sleep. Besides this, the eyes should be cleansed with warm water from all crusty exudations, and protected from the light by means of a large shade; this is better than closing them up with a handkerchief or bandage, as it allows the discharges to escape more freely from the palpebral sac.

When the acute condition has passed into a chronic state it must be treated as directed under the head of Chronic Catarrhal Conjunctivitis.

The treatment of an outbreak of ophthalmia.—The special steps to be taken to stamp out an outbreak of acute catarrhal conjunctivitis in a school, or any institution, must be thoroughly adhered to if success is to be attained; otherwise, if conducted in a slipshod manner, the ophthalmia will spread like wildfire with disastrous consequences.

(1) The whole institution must be examined, and all boys or girls with ophthalmia must be strictly isolated. On no pretext whatever may they be allowed to mix with the healthy.

(2) Each patient must have a separate towel strictly kept for his or her personal use. Any child found using another's towel or handkerchief must be punished. (Isolated and non-isolated.)

(3) A separate basin or tap to wash at must be reserved for each. (Isolated and non-isolated.)

(4) The swimming-bath must, for a time, be abandoned. It must be twice refilled and emptied before the non-isolated may have access to it.

(5) The non-isolated boys must be periodically examined, and any case with conjunctival discharge must be at once isolated.

(6) The isolated cases must be kept in a well-ventilated sanatorium with the light slightly reduced. Except with complicated cases, there is no reason why they should not have daily outdoor exercise in a part of the playground kept for their sole use. Lessons or work must be reduced or suspended, but the diet must not be altered nor any special privilege given them, otherwise self-inoculation will be attempted amongst those not isolated.

(7) The local treatment must be adopted as in sporadic cases (*vide supra*).

(8) No boy should leave the sanatorium without a certificate from the doctor in charge stating that he has been free from conjunctival discharge for a period not less than one week; he should then leave the school or institution for a period of one month in the country, and prior to mixing with the other boys (non-isolated) a second certificate of freedom from ophthalmia must be handed in. During his month's recruit his parents should be cautioned not to allow him to mix too freely with other children.

(b) *Follicular conjunctivitis* is recognised by the presence of small transparent elevations upon the lids and the culs-de-sac. These are associated with either acute or chronic catarrh, and according to the intensity of the inflammation so will their appearance be more or less modified. They are due to the abnormal development of the lymphoid follicles which, in health, are sparingly scattered in the subepithelial tissue of the conjunctival reflections. They are frequently grouped together, forming surface elevations which, though smaller than the large 'sago-grain' prominences of typical granular ophthalmia, are nevertheless easily visible to the naked eye. Besides these well-defined follicles, which are made up of lymphoid tissue and resemble in structure the solitary glands of the small intestine, we find microscopically a lymphoid cellular infiltration of the subepithelial tissue. This is distinctly marked off from the epithelial elements by the basement-membrane, as well as from the follicles by their vascular surrounding. The follicles may be situated deeply in the

fibrocellular tissue or immediately beneath the epithelium ; in the latter case they may or may not produce prominences on the conjunctival surface.

Etiology and pathology.—The cause of their unnatural increase is uncertain. The disease is more frequently found in youth than in adult life. It is very common in crowded dwellings, pauper schools, orphan asylums, and among those who dwell in a vitiated atmosphere. The xerosis bacillus has been discovered in this affection by Eyre,¹ though we are not in a position to attribute the disease to its presence. It is often produced in a mild degree by the prolonged use of eserine drops in cases of chronic glaucoma.

The *treatment* is the same as that for chronic catarrhal conjunctivitis. The symptoms are often relieved before the follicles have disappeared, but by persevering treatment these will sometimes take their departure without leaving any trace of their existence.

It will be seen under the head of Granular Ophthalmia how difficult it often is to make a positive diagnosis in these two affections.

2. **Purulent conjunctivitis** (purulent ophthalmia, blennorrhœa) is an acute suppurative inflammation of the conjunctiva, the outcome of inoculation with the gonorrhœal virus. For convenience of description and treatment it is divided into two classes—viz. gonorrhœal ophthalmia and ophthalmia neonatorum.

(a) *Gonorrhœal ophthalmia.*—*Symptoms.*—It commences about the third day after inoculation. The lids become swollen, red, glazed, and œdematous, so that the patient is unable to open the affected eye, and the medical attendant may be unable to do so without the use of a retractor (fig. 26). The conjunctiva of the lids and of the fornix is intensely swollen, and that of the globe is in a state of chemosis. In severe cases of acute catarrhal ophthalmia we have seen that the lids are sometimes much swollen, but here the infiltration often becomes so great that the lids are quite stiff and brawny ; they may be so thickened that they can only be everted by dividing the skin at the outer canthus. The lymphatic glands

¹ *Journal of Pathology and Bacteriology*, 1896.

immediately in front of the ear are often swollen and tender. The disease is very painful during the first two or three days. This is called the *period of infiltration*; the discharge at this time is chiefly composed of serum mixed with tears, and is sometimes even bloodstained; the swelling of the lids reaches its maximum. Next comes the *period of suppuration*, in which the discharge becomes thick and purulent, and the swelling of the lids begins to show signs of relaxation by the appearance of slight wrinkling of the skin. The ocular conjunctiva is densely infiltrated and vascular. It projects forwards as a fleshy wall around the cornea. In cases in which pannus has been treated by inoculation with gonorrhœal pus, the cornea also presents the appearance of a swollen fleshlike membrane. The pain now becomes decidedly less, and the swelling of the conjunctiva gradually diminishes. In favourable cases it returns to the normal in four or five weeks, whilst in the majority there follows a condition of chronic conjunctivitis similar to that already described as chronic catarrhal ophthalmia; the thickness and swelling of the lids having gone, and the purulent discharge having ceased, the conjunctiva is red, rough, velvety, and thickened, especially over the tarsus.

In the most intense cases the infiltration is sometimes so severe that the conjunctiva is no longer red, but assumes a greyish-yellow appearance, whilst a stiff, elevated, grey or grey-red wall is formed around the circumference of the cornea.

Mild cases of gonorrhœal ophthalmia sometimes occur, in which there is less pain, less infiltration of conjunctiva, less swelling of the lids, and even less discharge of pus than is described above. Such cases are often difficult to distinguish from acute catarrhal conjunctivitis (p. 65), and demand the most careful attention on the part of the medical attendant in order to arrive at a correct diagnosis. The most convincing proof of the gonorrhœal nature of the affection is the presence of the gonococcus of Neisser in the discharge; whilst its absence, as proved by thorough microscopic examination, is strongly in favour of the case being one of acute catarrh only.

The presence of the gonococcus is easily ascertained by the use of a $\frac{1}{12}$ inch oil-immersion lens. The examination is made as follows: Place a drop of the discharge on a cover-glass; place

a second cover-glass over this, and press them gently together so as to form a thin layer of pus on each ; then slide them apart and dry gently over a gas flame. Place a drop of a 2 per cent. alcoholic solution of methylene blue upon the cover-glass, and leave it for thirty seconds ; now wash away with care the excess of stain, dry as before, and mount in Canada balsam. It is now ready for microscopic examination.

The appearance of the organisms is shown in fig. 25. They occur mostly in pairs, and are somewhat kidney-shaped. They are found in the epithelial cells, in the pus cells, and in the fluid

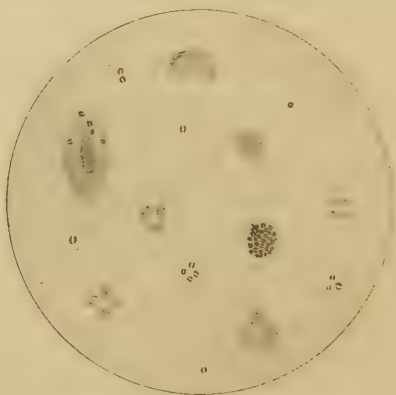


FIG. 25.—Gonococci.

containing these. They are exactly similar to the organisms found in the urethral discharge of gonorrhœa.

Complications.—The complications liable to accompany gonorrhœal ophthalmia are inflammation, ulceration, and sloughing of the cornea, iritis, and panophthalmitis. The cornea should be thoroughly examined every day by carefully separating the eyelids and raising the upper lid by means of a lid retractor (fig. 26).

a. *Corneal ulcers.*—The first sign of corneal trouble usually shows itself by superficial dulness ; this may be only partial or may occupy the entire surface. It next becomes more opaque, causing a milky-white or yellowish-white appearance. Ulceration now commences and extends deeply into the substantia propria of the cornea, so that, owing to the normal intra-ocular

pressure, perforation may soon take place, and the eye is thus placed in jeopardy. Ulceration often occurs near the circumference of the cornea, leaving the central portion free, or only hazy. It is better for perforation to take place here than at the centre of the cornea, as the latter may become clear after the attack has passed off. Sometimes, however, inflammation occurs all around the corneal margin, giving rise to a ring-abscess, and, by thus interfering with its nutrition, causes total sloughing.

The earlier in the course of the disease the cornea becomes affected, the greater is the danger of ultimate loss of sight; hence the importance of making a careful examination of the cornea each day after the onset of the disease. Should this become hazy or ulcerated during the first few days, it must be regarded as a sign of great danger. On the other hand, when ulceration makes its appearance only after the tenth day, it is less likely to cause total destruction of sight, and, with careful treatment, may not extend so deeply into the substantia propria as to cause perforation, but may heal up with very slight loss of tissue and even of transparency. These late-appearing ulcers occasionally occur without previous opacity; they generally occupy only a portion of the surface.

b. *Iritis* may occur without corneal trouble; it is usually present when the cornea is ulcerated, especially when perforation has taken place.

c. *Panophthalmitis*.—When the ulcer is large and occupies the central portion of the cornea, there is first a bulging of Descemet's membrane in the form of a pouch, into which the iris is pushed; then perforation takes place, and the iris protrudes from the aperture. Thus exposed to the purulent discharge, inflammation is immediately increased and conveyed to the ciliary body, the choroid, and all the remaining tissues of the eye, causing acute panophthalmitis. Sometimes even the crystalline lens and part of the vitreous body will escape from the corneal opening. After severe perforation and inflammation of this kind there is but little hope of restoration of vision, the corneal tissue which remains being usually opaque, and the iris being either clamped in the cicatrix or adherent to its posterior part.

Etiology and pathology.—The only cause of this terrible

disease is direct inoculation from the genitals or from a similarly affected eye, either of the patient or of another person. The pus may be conveyed by the finger, by pocket-handkerchiefs, by towels, or in other ways.

The actual poison is the gonococcus discovered by Neisser in 1878—a comparatively large diplococcus which is readily stained with methylene blue, in fact with all basic aniline dyes, but does not take Gram's stain, a point of great diagnostic value. It cannot be grown on any medium except human blood serum and egg albumen. Its growth is slow, and not till the lapse of three days is any visible alteration noticed in the medium. Though not anaërobic, it appears to grow more rapidly in the absence of oxygen. The cocci are found chiefly grouped in the interior of the pus cells, some in the epithelial cells, and others in the intercellular *liquor puris*. No animal has yet been inoculated, but true gonorrhœa has been produced in the vagina of a woman after the twentieth culture. Though a most destructive poison, it cannot be looked upon as very contagious, otherwise gonorrhœal ophthalmia would not be so rare. The discharge when dry, or if well diluted, is innocuous. I know of no instance of ophthalmia acquired by an individual from using a bath previously occupied by an infected person.

Treatment.—Prophylactic measures constitute not the least important part of the treatment. If one eye only is attacked, the other must be protected. Patients suffering from gonorrhœa should be warned of the danger attending the introduction of discharge from the genitals into the eyes of themselves and others, either directly by the finger or indirectly by means of towels, handkerchiefs, or other things. The medical attendants and others who may have charge of the patient should be most careful of their own eyes.

To protect the sound eye Buller's shield may be employed. This has the double advantage of giving the patient a certain amount of vision and of enabling the surgeon to examine the eye without disturbing its dressings. It is constructed as follows: Take a watch-glass and two pieces of very adhesive plaster, one about $4\frac{1}{2}$ in. square and the other 4 in. square; cut a round hole slightly smaller than the watch-glass in

the middle of each piece of plaster. Then insert the watch-glass between the two, and stick them together so as to form a small window. Now arrange the plaster by its free edge along the nose, forehead, and cheek, leaving a small space free to the outer side for ventilation.

The sound eye may instead be protected by clean lint or Gamgee tissue and a bandage, but in this case it should be examined daily and re-dressed at once.

In treating the affected eye, the indications are to cut short the inflammatory process, to reduce excessive pressure upon the globe by the swollen lids and retained secretions, and to treat any complications that may arise.

In the first stage—the stage of infiltration, before suppuration has set in—iced-water compresses of lint should be laid gently over the closed lids and changed at frequent intervals as soon as they seem to get warm. If pain and infiltration



FIG. 26.—Desmarres's Lid Retractor.

are severe, three to six leeches should be applied around the orbit. The palpebral sac should be frequently washed out with solution of corrosive sublimate 1 in 5,000; this should be done at least every hour. If the lids are so stiff and swollen that they cannot be separated by means of the lid retractors (fig. 26), the outer canthus must be divided. One blade of a pair of strong scissors being placed inside the sac and the other outside, all the structures are freely divided as far as the outer angle of the orbit. This gives rise to free hæmorrhage and escape of serum; it relieves the tension of the lids and their consequent pressure upon the globe; it often diminishes the pain, and it enables the surgeon not only to separate the lids and so irrigate the palpebral sac, but also to evert them and examine the palpebral conjunctiva and the upper and lower culs-de-sac. Bleeding should be encouraged by fomentation for a short time, and then the iced-water

dressings should be re-applied. After the cure of the ophthalmia the lids can be easily restored to their former condition by a canthoplastic operation, should they require it.

As soon as the *stage of suppuration* has set in, we use a 2 per cent. solution of nitrate of silver once in twenty-four hours in addition to the other local applications. The palpebral sac is first cleansed by irrigation with the sublimate solution; the lids are then everted and carefully dried with a piece of dry lint, and the nitrate of silver is applied with a small brush and allowed to remain for about a minute, when it is washed away with sublimate solution and the lids gently restored to their normal position. The condition of the cornea should be carefully examined at this time, and a drop of atropine sulphate solution ($\frac{1}{2}$ per cent.) applied in order to keep the pupil dilated, in case of iritis.

The duration of this kind of treatment varies with the nature of the case: it may have to go on for ten to twenty days. In slight cases, where swelling, pain, and suppuration are not excessive, the irrigation with sublimate and the daily application of the nitrate of silver may suffice to cure the affection in a comparatively short time.

When the *third or chronic stage* is reached—that is, when the swelling, suppuration, and pain have departed, leaving only a velvety or granular-looking thickened palpebral conjunctiva and a muco-purulent discharge—the case must be treated with milder astringents in the same way as a case of chronic catarrhal conjunctivitis.

Treatment of complications.—The most serious complication in gonorrhœal ophthalmia is, as we have just mentioned, ulceration of the cornea. It behoves us, therefore, to be very careful and gentle in the manipulation of the eye and the eyelids, during the necessary and numerous examinations, not to cause any abrasion of the corneal epithelium and so favour the entrance of the virus into the deeper layers of the cornea. For this reason irrigation by means of cotton-wool or lint is preferable to the use of a syringe. For the same reason, when a slight ulcer is found to exist we must be careful not to aggravate it; and when an ulcer has proceeded to the point of perforation this carefulness is all the more necessary.

In very severe and extensive ulcers almost all treatment is powerless to prevent the destruction of the corneal tissue.

When *ulcer of the cornea* exists, the treatment of the case requires to be modified. The eye must be lightly bandaged so as to give support to the cornea through the lids. The bandage must be removed at intervals and the irrigation with weak sublimate solution performed. Atropine and cocaine solution may then be dropped in ; it will dilate the pupil and soothe pain. If suppuration is abating, it would be well now to discontinue the daily use of nitrate of silver to the lids , but if not, this must be continued, great care being exercised not to allow the application to extend to the cornea. Iodoform ointment, 1 per cent., may be placed in the palpebral sac. Iced-water compresses had better be substituted by warm fomentations every few hours. If a perforation should occur near the margin of the cornea, eserine drops may be used instead of atropine, with the view of contracting the pupil and so preventing prolapse of the iris ; if perforation is central, the atropine should be continued with the hope of keeping the iris out of the central opening ; this, however, is rarely effected, especially where the perforation is at all extensive.

Besides these medical remedies for the complications of ulcer of the cornea, we must also resort to operative treatment. This should commence early in all severe cases, and should not be delayed even in small ulcers when these do not quickly respond to the medicinal remedies which have just been described. The best and most reliable way of checking the rapid inroads of the suppurating ulcer is by the galvanocautery.

This small eye-cautery should be used at a dull red heat. The patient should be placed under chloroform, as in this inflamed condition the cornea does not become fully anæsthetised by cocaine. The cautery should be thoroughly applied to the whole floor of the ulcer and made to touch the edges of the healthy tissues around. The object of the cauterisation is twofold—first to destroy as far as possible the infecting virus which has entered the corneal tissue, and secondly to give free outlet to the pus which has formed. The instrument thus used may perforate the cornea. If hypopyon already

exists, this may be desirable, but otherwise it is better, if possible, to leave Descemet's membrane intact, and so prevent protrusion of the iris and other possible consequences of perforation.

The older method (Saemisch) of incising the abscess or ulcer quite through its base and into the anterior chamber is more likely to produce the sequelæ just mentioned ; although, failing cauterisation, it liberates the pus from the cornea, and so tends to prevent that total destruction of tissue which, without some aid of this kind, so frequently takes place.

After the operative treatment, the local medication is continued as before mentioned.

(b) *Ophthalmia neonatorum*, or purulent conjunctivitis occurring in the newly born, is identical with that just described as gonorrhœal ophthalmia.

Cause.—In the passage of the child's head through the vagina the eyelids become smeared with vaginal and urethral secretion, and as soon as the eyes are open they can easily become inoculated with any gonorrhœal virus that may be contained in the discharge ; or, supposing the discharge to be free from gonorrhœal poison, it is equally possible for an acute catarrhal conjunctivitis to be set up in the same way. It is remarkable that a very slight and old-standing gonorrhœal discharge will set up a very acute gonorrhœal ophthalmia either in the eyes of the newly born or in those of adults. The affection usually declares itself about the third day, rarely on the fourth or fifth day. Should it come on at a later date, it can no longer be attributed to the birth, but some other means of inoculation must have been in operation.

There is a form of this disease which is termed *ante-partum ophthalmia*, of which there are several well-authenticated cases. In some instances, infection took place in protracted labour, the membranes having ruptured forty-eight hours before the delivery of the child. In others, the time was short, and the explanation must be either that the micro-organisms passed through the membranes before they ruptured, or that in these cases the gonococci were exceptionally virulent.

Its *symptoms* and *complications* are essentially the same as those occurring in gonorrhœal ophthalmia, except that they

are somewhat less pronounced in degree. The swelling of the lids is less hard and brawny ; the flow of pus is less copious ; the ocular conjunctiva is not so much affected, as shown by the absence of any extensive chemosis. The cornea is less liable to inflammation, abscess, or ulceration. If ulcers appear, as they frequently do in neglected cases, they are less rapid and destructive in their course.

As in the case of adults, so here we have to diagnose between a merely acute catarrhal ophthalmia and a virulent affection arising from inoculation with gonorrhœal pus from the genitals of the mother. This can only be done by careful microscopic examination of the discharge, as described on p. 71.

If gonococci are absent, we are justified in concluding that the case is not one of gonorrhœal origin, and, therefore, is neither dangerous nor difficult of cure.

Treatment.—As with adults, so with the newly born, the most scrupulous and diligent attention should be given to prophylactic measures. In whatever station of life our patient may be, it is always a safe preventive against this form of ophthalmia to thoroughly disinfect the vagina by injections of sublimate solution 1 in 1,000 during the act of parturition. Secondly, the eyes of the child should be thoroughly cleansed, if possible before they are opened, with clean lint. During the first bath the eyes of the child should not be exposed to the bath water ; but as soon as the rest of the body is cleansed, and the child wrapped up, the eyes should first be bathed with clean water and a special towel, and then a drop of 2 per cent. solution of nitrate of silver should be instilled into each eye. If this method, which was introduced by Credé, were carefully practised, this frequently occurring and dangerous affection would be seldom seen.

Both eyes are usually attacked in children ; but if only one is affected, the Buller's shield is hardly applicable and not necessary. The other eye should, however, be closed and secured from infection by lint, cotton-wool, collodion, and strapping. It should be examined once daily.

The treatment of ophthalmia neonatorum is the same in principle as that for adults, but, owing to the tender age and

delicacy of the subjects, it requires a few remarks as to detail. The child should always be seen at least once in twenty-four hours. In order to examine the eyes, the surgeon, being seated and in a good light, directs the nurse to place the child's head between his knees, which are protected by a towel. He then first cleanses the eye by irrigation with tepid solution of corrosive sublimate 1 in 5,000. He next everts both eyelids and cleanses them; dries them with soft lint, and applies a 2 per cent. solution of nitrate of silver to their surfaces; the excess of this is then washed off and the lids closed. Having done this, he separates the eyelids by means of two retractors (fig. 26) in order to examine the cornea. Should this be at all cloudy, a little atropine, $\frac{1}{2}$ per cent., must be dropped in to dilate the pupil in case of iritis. Should ulceration of the cornea be found to exist, it must be treated on the same lines as have just been described for adults (p. 77), but always bearing in mind the greater delicacy of the young organism. Before suppuration has set in, simple and thorough cleansing of the palpebral sac with the sublimate solution and cold compresses of the same is sufficient. After suppuration has commenced, the daily application of the 2 per cent. silver-nitrate solution must be added. In these cases the latter should be persevered with until complete cure is effected, otherwise the disease is apt to relapse. In a case of excessive suppuration it is sometimes well to apply the nitrate of silver twice daily. The object of this apparently severe remedy is germicidal. The caustic destroys the superficial layers of epithelium, which are immediately cast off, and so enables the silver and the sublimate to act upon the deeper layers of the tissue into which the virulent germs have penetrated. In sections of the mucous membrane in these gonorrhœal affections the gonococci may be seen abounding in the deeper layers of the epithelium. If we find the cornea clear in a case of ophthalmia neonatorum, at whatever stage, we may safely prognosticate that by proper attention to the above details no bad results will occur.

In those exceptional cases where no gonococci can be found in the discharges, and which therefore, as already mentioned, need not give so much anxiety, the same treatment can be

employed as in the virulent cases. They will yield much more quickly to treatment.

3. **Granular conjunctivitis** (trachoma, granular ophthalmia, Egyptian ophthalmia, military ophthalmia) is a disease characterised by the presence of granular elevations in the culs-de-sac and, to a less extent, on other parts of the conjunctiva, attended with inflammatory changes and subsequent cicatricial contraction.

This complaint may be *acute* or *chronic*. The latter form is far more frequently met with in this country, and presents great variations in kind.

Acute granular ophthalmia is recognised by the presence of semi-translucent granules in the culs-de-sac of the conjunctiva. This is the only distinctive feature between it and acute catarrhal ophthalmia: when this is not pronounced it is impossible to distinguish the two affections. The granules are not usually seen at this early stage on the palpebral conjunctiva, as they will be when the disease lapses, as is only too probable, into the chronic form. The resistance to treatment and eventual chronicity will suggest the nature of the affection we have to deal with.

Symptoms.—There are more or less copious muco-purulent discharge, intense photophobia, with a gritty feeling as of sand in the eye; lachrymation is marked, the hot tears gushing out when any attempt is made to separate the lids. Exposure of the eye or painting the lids often produces a paroxysm of violent reflex sneezing. Upon everting the lids and examining the culs-de-sac (see p. 62), the conjunctiva will be found intensely injected, the rugæ in the fornices present a granular appearance, and flakes of lymph will be seen caught between the folds of the membrane. The small 'sago-grain' granules are seen most distinctly on the summits of the rugæ; they are devoid of vessels, and so appear translucent. The plica semilunaris and caruncle are red and swollen, and chemosis may be present. As a rule, after a week or fortnight the symptoms gradually subside, the inflammatory changes become less marked, and a simple form of chronic trachoma remains. Complete resolution is sometimes seen, especially in those countries where acute trachoma is common, as in Egypt and in the woods of Southern

Australia. In some instances, acute trachoma continues with unabated severity for weeks or months, resisting all treatment; this class of cases will come under the heading of the malignant variety of chronic trachoma (see below).

Chronic granular ophthalmia may follow an acute attack, or be chronic and almost devoid of all inflammatory changes from its onset. It is convenient to classify chronic trachoma into three chief groups:

(a) *Slight forms*, in which there is slight redness of the free edges of the lids, a feel of grittiness in the eyes, and an increase in the secretion of mucus. On everting the lids, however, we find fine granulations disseminated over the conjunctiva, mostly



FIG. 27.—Everted Granular Lids.

in the position of the upper and lower culs-de-sac. The conjunctiva over the tarsi is often free, or the granulations may be seen creeping over their borders near the canthi.

(b) *More severe forms*, in which there is granulation and infiltration of the conjunctiva of the lids as well as that of the culs-de-sac. The mucous membrane is thickened, and presents a greyish rough appearance, being studded with grey or yellow translucent roundish granules. The edges of the lids are reddened; there is much secretion of mucus, which is often semi-purulent. Lachrymation and photophobia are always present, and in some cases are very troublesome.

(c) *Malignant*.—This third group may be considered the perpetuation of acute trachoma. It is a still more grave and

troublesome class of cases. The inflammatory signs are very marked, and the granular affection extends to the ocular conjunctiva, and even to the cornea. The palpebral conjunctiva and fornices are much infiltrated and thickened, and present fleshy villous-looking growths, which bleed on the slightest touch. The whole episcleral and corneal surfaces become filled with tortuous blood-vessels, and the cornea becomes quite cloudy and vascular—*pannus*. There may be superficial, deep, or even perforating ulcer of the cornea.

The most discouraging feature of this malignant form of trachoma is its obstinate resistance to treatment. The inflamed tissues do not return to their normal state, but appear to undergo an ultimate fibroid degeneration. The mucous membrane becomes thin, shrunken, and tightly adherent to the tarsi; the culs-de-sac are obliterated, and the tarsi become shrunken and incurved—*entropion*. This condition must not be confused with the essential shrinking and other symptoms attending pemphigus of the conjunctiva.

Etiology and pathology.—The chief cause of this affection is contagion. This view is substantiated by the fact of its prevalence in pauper schools in past and even in present times; also in prisons, barracks, and other places where there are crowded communities, having facilities for the conveyance of unhealthy secretions from eye to eye by means of towels and otherwise.

No race is immune. In general, the higher altitudes are unfavourable to the spread of trachoma. Of all races, the Celts are the most resistant. The Irish, Italians, Jews, Poles, and Japanese are especially liable to be attacked. The Negro races are almost immune. In foreign climes, as Egypt, Australia, and the like, where the disease is rampant, the flies are the chief agents in spreading the complaint; and, no doubt, from the simple conjunctivitis set up by dust, intensity of light, &c., the conjunctiva is placed in such a condition that it forms a suitable *nidus* for the growth and development of any infecting organism. The disease is probably caused by a pathogenic micro-organism, which alone can furnish us with a scientific diagnostic proof. A diplococcus has been described by Sattler to which he attributes the disease. It is much smaller than, though resembling, the gonococcus, and is not

decolorised by Gram's staining process. It has been grown on agar and blood serum. There has been much dispute about this coccus ; and we must, for the present, admit that the pathogenesis of trachoma still remains in obscurity. In all cases, whether produced by contagion or otherwise, the subjects of the affection appear to have been predisposed to it by ill-feeding, over-fatigue, bad ventilation, and other debilitating causes. It is rarely seen in the better classes of society.

This disease appears in the form of numerous small greyish, hemispherical, semi-translucent elevations, having a great resemblance to boiled sago-grains (follicular granulations). These are seen first in the fornices, and, later, on the palpebral conjunctiva.

Microscopically, these granular elevations are usually found to be non-vascular lymphoid follicles situated immediately beneath the epithelium. They are composed of a circumscribed collection of lymph-corpuscles, with a delicate reticulum. Surrounding each follicle is a capillary network of blood-vessels, which separates them from a diffuse vascularised lymphoid tissue, in which they are imbedded. The surface epithelial cells of the conjunctiva are disturbed by the migration of these lymph-corpuscles between them, and the outline of the basement-membrane becomes completely lost. Many of the epithelial cells become vacuolated, some distended with mucin, and others, according to Ridley, invaded by sporozoa. Ingrowths of the epithelium may take place and form simple tubular glands, lined by columnar and goblet cells which secrete mucin. This occurs in all chronic inflammations of the conjunctiva.

Though lymphoid follicles are usually found in excess in the conjunctival membrane during an attack of trachoma, they are not essential, for in some forms of granular ophthalmia the granulations are caused by projections of diffuse vascularised lymphoid tissue, without the presence of a single follicle. On the other hand, a proof of their new formation in this disease is illustrated by a case under my care in which I had the opportunity of examining the cornea, vascularised and disorganised throughout by a severe and progressive pannus. Imbedded in the cellular new-growth which occupied the place

of the substantia propria, I found well-marked circumscribed and non-vascular lymphoid follicles exactly like those found in the retrotarsal folds of similar cases of trachoma. Beneath these follicles, in the deeper layers of the conjunctiva, numerous vessels are seen traversing fibrocellular connective tissue. Around the vessels are aggregated numerous formative or epithelioid cells indicative of developing fibrous tissue. After the follicular granulations have existed some time, the adjacent papillæ become hypertrophied, and the whole lid assumes the rough villous appearance which is so often left after catarrhal or purulent conjunctivitis—papillary granulations (see fig. 27). As time goes on, the connective-tissue element increases, and converts the conjunctiva into a dense fibrous structure. The culs-de-sac become obliterated and the eyelids deformed; the movements of the globe also are interfered with by this universal cicatricial contraction of its enveloping mucous membrane.

From what has been said with regard to the pathological anatomy of this affection and follicular conjunctivitis, it is evident that, by the aid of the microscope alone, a distinction is difficult. The scantiness of the diffuse lymphoid tissue, and the well-defined basement-membrane to the epithelium in the latter affection, are points of difference worthy of mention. The main distinctive features are clinical.

Complications and sequelæ.—As complications, ulceration of the cornea and pannus are most common. Marginal blepharitis may be caused by the irritating discharge; spasmodic entropion of the lower lid from persistent blepharospasm. As sequelæ, cicatricial entropion of the upper eyelid, trichiasis and nebulous condition of the cornea, are more frequently seen. The palpebral fissure, from the general cicatricial contraction of the conjunctiva, is rendered smaller (blepharophimosis), and cannot be opened to such a wide extent as formerly.

Treatment must in all cases be general as well as local. The *general treatment* consists in placing the patient under the best possible hygienic conditions. Good and plentiful nourishment, exercise in the open air, and well-ventilated sleeping accommodation are essential adjuncts to local treatment. Change of air, or a sea voyage, is frequently of great

assistance. The eyes should be protected from bright light and from dust by smoked glasses. The patient should avoid as far as possible over-fatigue of the eyes, especially by artificial light. Tonics, such as iron, quinine, cinchona, and cod-liver oil, should be administered. Parrish's food, Easton's syrup, and similar forms of medicine, are beneficial.

The *local treatment* must have the twofold object of, first, reducing the inflammation and its accompanying copious and infectious secretion; and, secondly, of destroying or eradicating the granular infiltration of the conjunctiva and subconjunctival tissues.

Apart from surgical interference, the treatment resolves itself into the daily application of caustics, the chief of which are the nitrate of silver in 2 per cent. solution and the sulphate of copper in crystal. The *nitrate of silver* is less irritating than the blue-stone, and is indicated in all recent cases attended with inflammation and copious purulent or semi-purulent secretion—indeed, in any case, whether old or recent, where purulent discharge is present, it is the better agent to employ. The lids must be well everted and the culs-de-sac exposed, their surface being dried with lint before the application, and the excess being washed away after an interval of one or two minutes by water or 1 per cent. solution of common salt. This prevents the brown staining of the sclerotic which is sometimes produced.

The *sulphate of copper* crystal is indicated in all cases where inflammatory signs are slight and there is much hypertrophy of the diseased parts. Its action is much more irritating than that of the silver, but it is more efficient in reducing the hypertrophy. It is not tolerated where there is much inflammation.

It will thus be seen that nitrate of silver is indicated until the secretion is reduced and the inflammation diminished, after which it can be advantageously replaced by the blue-stone. One application daily is sufficient for all but severe cases, when it is better to apply it twice. It should also be applied energetically—*i.e.* with some pressure and rubbing—in severe cases, and lightly in slight ones. The process is a very long and tedious one, extending possibly over months or even years. It must be continued regularly until all traces

of the affection have disappeared, and must be renewed immediately if the affection crops up again, as it is always liable to do. Should the cornea be ulcerated, care must be taken to keep these caustics as much as possible away from the ulcer, and to wash the excess well away from the palpebral sac after each application. These reagents may be supplemented by cold compresses of boric acid 2 per cent., or sublimate solution 1 in 5,000.

Owing to the tedious nature of the treatment, it is well to teach a friend of the patient to evert the lids and apply the blue-stone.

Reference has been made elsewhere to the treatment of pannus by producing jequirity ophthalmia.

The classical treatment just described, however, is wearisome alike to the patient and to the medical attendant. The patient is usually poor and unable to attend regularly for treatment; he consequently is often obliged to suffer for months or years, and finally to find himself the subject of shrunken conjunctiva, entropion, pannus, or even loss of sight. This being the case, more active measures have been tried with a view to a more rapid and radical cure: the galvano-cautery, electrolysis, excision of the culs-de-sac, scarification, scraping, expression, and other means have been tried with considerable success. The *galvano-cautery* applied to the granulations once a week, and combined with the caustic treatment above mentioned, I have found a very beneficial adjunct.

Darier's method.—Better than this, however, is the surgical procedure introduced by Darier, consisting of free scarification of all the affected surfaces, followed by scraping with a sharp spoon, brushing away of the gelatinous infiltration with a small hard toothbrush steeped in 1 per cent. solution of perchloride of mercury, and final washing with a solution 1 in 1,000 of the same.

This is a severe method of procedure. It requires the administration of a general anæsthetic, and produces considerable reaction; but it is followed by marked improvement within the first few weeks, and the disease is usually almost exterminated in from one to three operations.

It is applicable to almost all cases of trachoma—exception

being perhaps made in violently acute cases, where there are excessive redness and tumefaction of the whole conjunctival surface, œdema of lids, and copious discharge. Here it is well to modify the operation until the acute symptoms have become reduced. This will be effected by scarifying the conjunctiva and applying solution of nitrate of silver (2 per cent.) immediately after the operation, and continuing to apply it once daily. When the extreme inflammatory symptoms have subsided, recourse may be had to the more radical cure above referred to.

Before commencing the operation, a careful examination of the entire conjunctival surface must be made, so that the exact distribution of the affected surface can be made out. If this precaution be not taken, it will be less easy to do so when the operation has commenced, owing to the hæmorrhage, which is often copious. It is most important that the whole affected area should be attacked, so that every possible 'trachomacoccus' shall be either removed or destroyed.

In order to thoroughly evert the upper lid and expose the upper cul-de-sac, a pair of thin-bladed dressing-forceps may be used. These are inserted at the outer canthus so as to grasp the lid parallel to its ciliary border. Sattler has introduced some spikes to this, so as to form a male and female blade, which renders the lid more secure. Should there be difficulty in everting the lid, it is better to divide the outer canthus with scissors. Very little harm is done by this incision: it usually comes right of itself, or can be afterwards rectified by a small operation. Should the caruncle show signs of infiltration, it may be excised with scissors. Should the cornea be affected with fleshy pannus, it may be gently scraped and brushed from the centre towards the periphery.

It is well to commence the scarification, scraping, and brushing upon the lower lid and cul-de-sac, so as to avoid the hæmorrhage which would proceed from the upper parts. The lid must be seized with the forceps about 2 mm. from its margin, and well everted by one or, if necessary, two turns. The scarifications are then cautiously made so as to divide and open up the granulations—a sharp scalpel or a cataract knife will suffice for this. The depth of the incisions must be proportionate to that of the infiltration. The scarification is followed by gelatinous exudation, which must be wiped away with a piece of clean lint.

Next, a sharp spoon is used to scrape the incised surface and so get away or loosen the diseased tissue. After this, a small tooth-

brush with short stiff bristles is steeped in 1 per cent. solution of sublimate and used moderately vigorously over the surface which has been incised and scraped. The forceps are then removed, and the conjunctival surface which they covered is similarly treated. Next, the upper lid and upper cul-de-sac are attacked in the same way, it being remembered that the upper cul-de-sac is the principal habitat of the disease. The incisions here require to be rather deeper than in the lower parts. After these parts have been duly scarified, scraped, and brushed, the whole conjunctival surface is carefully washed with sublimate solution 1 in 1,000. A few drops of atropine and cocaine solution are instilled into the palpebral sac, and the eyelids are closed and kept cool by cold or iced compress of perchloride solution 1 in 5,000.

The whole conjunctival surface must be examined once daily after the operation, and cleansed by cotton-wool steeped in warm perchloride solution 1 in 1,000.

This operation is somewhat heroic, and would appear calculated to destroy the whole conjunctival surface. It is, however, less destructive of that membrane than the disease; the long strips of conjunctiva appear to grow rapidly, and the surface soon appears to be intact again. Of course, great care must be taken to separate the opposed surfaces each day after operation, or symblepharon is liable to ensue.

Hotz's operation, or the operation of expression of the granulations, is held in great repute by many ophthalmic surgeons. It essentially consists of squeezing out the granulations by the pressure of forceps. Numerous forceps have been specially devised for this operation by Grady, Knapp, Stephenson, and others. The conjunctiva being well cocainised, and cleansed with sublimate solution, the upper eyelid is everted, and one blade of the forceps is inserted into the cul-de-sac and the other on the palpebral conjunctiva; then with steady and firm pressure the forceps is drawn away, expressing the granulations. The whole conjunctiva, where diseased, is submitted to this expression process. The lower eyelid is more difficult to deal with; it is easier to place one blade on the



FULL SIZE

FIG. 28.—Knapp's Roller Expression Forceps.

cutaneous surface and the other in the cul-de-sac. There is considerable bleeding, and the granulations may be carried by the force some distance, so it is advisable to wear protective glasses while performing the operation. A general anæsthetic is sometimes necessary, as it is a painful process. The eyes should not be bandaged, but bathed frequently with sublimate lotion.

4. **Membranous conjunctivitis** is comparatively rare in this country; nevertheless, a good number of cases have been recorded; and in Germany, where the graver forms of the affection appear to be of more frequent occurrence than in Great Britain and France, the subject has received considerable attention.

Von Graefe endeavoured to arrange these cases into two classes—viz. croupous and diphtheritic; and, for convenience of description, this method is adopted here, although intermediate cases occasionally present themselves.

(a) *Croupous or pseudo-membranous conjunctivitis* is characterised by the appearance of a whitish-grey membrane occupying the conjunctiva of one or both the eyelids, and sometimes that of the globe or culs-de-sac also. This adheres somewhat tightly to the subjacent tissue, but involves only the epithelial lining, and can be removed with forceps. The surface beneath is found to be very red and perhaps bleeding here and there. The exact nature of this affection is obscure, and whilst some cases are attended with considerable inflammation, especially at the onset of the disease, others appear to be quite chronic and persistent. The treatment consists in removing the fibrinous exudation and stimulating the subjacent surface with 2 per cent. solution of nitrate of silver daily. Should inflammatory symptoms be very troublesome, they may be treated as in catarrhal ophthalmia. The cornea is seldom affected.

(b) *Diphtheritic conjunctivitis* is similar in nature to diphtheria of the throat or other membranes, and, like them, is accompanied by fever and great prostration. It is peculiarly dangerous to sight, owing to the rapid destruction of the cornea which it frequently produces; it may even cause death. It most commonly occurs in children under twelve years of age.

Symptoms.—In severe cases, the first signs are those of catarrh; the lids are red, swollen, hot, and painful. After twenty-

four hours, however, the swelling becomes considerably greater : the lids are so stiff and brawny that they can hardly be separated so as to expose the cornea. The conjunctiva also is much thickened, deeply congested, and shows isolated areas of a pale greyish-yellow colour in which ecchymotic patches are visible. These patches are diagnostic of the disease, and indicate that exudation is taking place into the deeper parts of the membrane, leaving it pale and bloodless. There is also a thin milky discharge which is extremely infectious. This condition—*the stage of infiltration*—continues for five to ten days, by which time the whole surface is often covered by and infiltrated with a greyish membrane, which cannot be peeled off with forceps, but tears away in shreds.

This is followed by *the stage of suppuration*, in which the brawny swelling of the lids disappears, and they are softer and more elastic. The discharge becomes purulent, the exudation is partly thrown off, leaving the conjunctiva in a sloughy and granular condition. The cornea, also, is usually involved in the process ; it has probably lost its transparency, and may have become partly or totally necrosed. The necrotic tissue is then gradually cast off ; the discharge diminishes, and *the period of cicatrisation* sets in. In severe cases this is attended with perforation and destruction of the cornea, shrinking of the conjunctiva, and symblepharon.

In lighter cases all the above symptoms are modified in degree, although they pass through similar stages of infiltration, suppuration, and contraction.

This disease may be mistaken for purulent or severe catarrhal ophthalmia, especially at its commencement or during the suppurating stage. It differs from these, however, (1) in the great stiffness of the lids, and its long duration ; (2) in the smooth yellowish non-vascular patches of the conjunctiva covered with small ecchymoses ; in purulent ophthalmia there is regular redness and succulent swelling of the mucous membrane ; (3) in the presence of fibrinous infiltration in the depths of the tissue, rendering it adherent, whilst any membrane existing in purulent cases can be easily removed ; (4) in the absence of the Neisser gonococcus and the presence of the Klebs-Loeffler bacillus in the discharges ; (5) in glandular enlargement, though this is some-

times present in purulent ophthalmia ; and (6) in the presence of constitutional disturbance, and the existence of albuminuria.

Etiology and pathology.—Similar to purulent, catarrhal, and granular ophthalmia, this disease is also the result of inoculation, and is likewise contagious. The poison is the Klebs-Loeffler bacillus. It resembles the tubercle bacillus in length, but is twice its breadth, and often presents club-shaped extremities. It grows rapidly on Loeffler's blood serum, *within twenty-four hours*, at incubation heat ; and is best stained with Loeffler's alkaline methylene blue. Associated with these organisms are invariably found pyococci, especially the streptococcus pyogenes. The constitutional disturbance is caused, as in the case of throat diphtheria, mainly by the absorption into the system of the toxine generated by the ferment action of the specific organism in the conjunctival membrane, in part also by the entry of the streptococci into the blood.

Treatment.—Directly the diagnosis is made (and all suspicious cases should be examined early for the pathogenic organism), constitutional treatment should at once be begun by the daily injections of the antitoxin.¹ Two may be sufficient. Rest in bed, liquid diet, and attention to the bowels, &c., are also necessary.

Locally, prophylaxis is of the first importance. If only one eye is attacked, the other should be protected as in purulent ophthalmia. Children, who are most liable to be attacked, should be kept away from the possibility of infection. During the stage of infiltration the eye should be carefully cleansed with weak antiseptic lotions ; where there is great swelling, with heat and pain, these may be applied in the form of iced compresses. Atropine should be instilled, with the hope of keeping the pupil dilated. Towards the end of this stage warm fomentations may be substituted for the iced compresses, so as to favour absorption of exudation and separation of sloughs.

¹ The dose of the antitoxin supplied by the British Institute of Preventive Medicine varies from 10 c.c. to 20 c.c. It is supplied in 30-c.c. phials. It is necessary to have an iridium needle to the hypodermic syringe so as to easily sterilise it.

When the stage of suppuration has set in, weak astringent lotions may be used, but caustics must be avoided.

The cornea must be carefully watched during the whole process, for upon its condition depends the future visual power of the eye. Should it be found to be gangrenous in its entirety, there is little to be hoped for; if only partly ulcerated, it must be treated according to the rules laid down in the chapter on Corneal Affections.

5. *Phlyctenular conjunctivitis* (also called *pustular*, *scrofulous*, and *strumous conjunctivitis*) is characterised by the presence of one or more small vesicular-looking bodies on the sclerotic portion of the conjunctiva (see fig. 7, opposite p. 118). Each is at first small, conical, and well defined; it seldom measures more than from 1 to 2 mm. across the base. It is at first transparent, but soon becomes yellowish, indicating the formation of pus. Sometimes it becomes firm in texture, forming a somewhat hard prominence. The surrounding conjunctiva is swollen and injected, and there is frequently a triangular leash of enlarged blood-vessels, having its apex at the phlyctenula and its base towards either the inner or the outer canthus. The number of these phlyctenulæ varies from one to five or six. One or two will appear by preference at the sclero-corneal junction, although they may be entirely corneal, or entirely in the sclerotic portion of the conjunctiva, or they may occupy any of these positions simultaneously; when, however, more than two occur, they generally appear in successive crops. So long as the corneal portion of the conjunctiva is not simultaneously affected there is little or no inconvenience beyond a pricking sensation, increased secretion of mucus, and more frequent blinking than normal. As soon, however, as the cornea is attacked (*phlyctenular keratitis*), even though it be near the periphery, there is increased lachrymation, and photophobia may be so great as to cause blepharospasm. In some cases these pustules are accompanied by a more extended inflammation of the conjunctiva, presenting the combined symptoms of muco-purulent and *phlyctenular conjunctivitis*.

Etiology and pathology.—The actual cause of this conjunctival eruption is not known. If unattended with discharge,

it is not contagious. It is more liable to occur in the children of tubercular parents, especially children who present the appearance of the so-called 'scrofulous diathesis.' It is curious how frequently it is associated with pediculosis of the scalp and contagious impetigo. These are merely evidences of neglect, and may have no causal relationship. Phlyctenular conjunctivitis is common in children up to the age of ten or twelve years, but may occur at any period of life. The subjects are generally anæmic, badly nourished, and live in crowded and ill-ventilated dwellings.

Prognosis and treatment.—So long as the corneal conjunctiva is unaffected, the phlyctenulæ break down after a few days, leaving a superficial ulcer, which rapidly heals, and the conjunctival redness disappears. The disease, however, shows a great tendency to recurrence.

In the early stage, or if there is absence of irritation, calomel powder may be flicked into the palpebral sac and on to the ocular conjunctiva with good results. If there is much irritation this must be avoided, and substituted by the yellow oxide of mercury ointment (F. 39) with, if necessary, a little of the alkaloid atropine dissolved in it. Boric acid lotion (F. 28) may also be prescribed if there is any discharge.

Constitutional treatment is also important. A wholesome diet and good hygienic conditions should be prescribed; also plentiful exercise in the open air, and the internal administration of tonic medicines—Parrish's food, cod-liver oil, iron or cinchona, &c. A visit to the seaside is also of service.

6. Spring catarrh (*Printanière, Frühjahrskatarrh*), described by Saemisch, is an affection of the conjunctiva very rare in Great Britain, but sometimes seen in young male subjects. It is characterised by the presence of large papillæ or granular elevations over the tarsi, generally of the upper lid; these are somewhat flattened on the surface, and they are not of the semi-transparent appearance of boiled sago-grains, but more like the reddened conjunctiva in which they are seated. It is very chronic in its nature, extending over several years and liable to recur, especially in the spring or early summer. It is not very painful, but is liable to be mistaken for trachoma.

The *symptoms* are slight intolerance of light, lachrymation, very little, if any, mucous discharge, and a peculiar drooping of the eyelids. Occasionally the ocular conjunctiva becomes reddened, and at the limbus near the outer and inner edges of the cornea there appears a reddish-brown, vascular, gelatinous-looking growth. The cornea is seldom affected.

Etiology and pathology.—Very little is known as to the cause of this affection. The thickening of the conjunctiva is mainly due to epithelial hyperplasia, and, according to Knies, it is a warty growth analogous to the common cutaneous lesions. It certainly is not a catarrh.

The *treatment* consists in allaying the irritation by cocaine and using slight astringents, as in chronic catarrhal conjunctivitis. I have found the galvano-cautery, freely used every one or two weeks, very efficient in reducing the large granular-looking elevations.

OPHTHALMIA NODOSA.

Ophthalmia nodosa (pseudo-tuberculosis of the conjunctiva—Pagenstecher) is a very rare disease of the conjunctiva, caused by the hairs of a caterpillar, the *Bombyx rubi*. The hairs penetrate the conjunctival tissue, and cause by irritation small nodules or tubercles to develop. There is generally a history of a caterpillar having been thrown at the patient. The nodules appear a few weeks after the injury, and are not necessarily limited to the conjunctival membrane. Graver troubles, keratitis or irido-cyclitis, may show themselves. A complete and interesting account of this affection is reported by Lawford.¹

TUBERCULOSIS OF THE CONJUNCTIVA.

Tubercular disease of the conjunctiva has been known for the last twenty-five years, but, as a primary disease, has only recently received its share of attention.

Varieties.—There are at least three distinct clinical varieties of conjunctival tuberculosis—tubercular ulcers, tubercular growths, and lupus of the conjunctiva. (a) *A tubercular*

¹ *Ophth. Soc. Trans.* vol. xv. p. 210.

ulcer appears, as a rule, on the palpebral conjunctiva, and may simulate a palpebral chancre, especially if near the margin of the eyelid. The ulcer is round, oval, or, by the coalescence of two or more, irregular, and about the size of a split pea; its surface is pink or pale yellow in colour, finely granular, and not indurated at its base. The pre-auricular gland on the same side is enlarged, firm, always tender, and sometimes painful. The submaxillary glands of that side may also be enlarged. It may be the source of general tuberculosis. Beyond slight discharge, redness of lid, and a certain amount of discomfort, the symptoms are not pronounced. (b) *Tubercular growths* of the conjunctiva have almost a characteristic appearance. There are definite red cock's-comb growths projecting from the fornices and from the palpebral conjunctiva. The whole membrane is much thickened and rugose. These papillary outgrowths are seen to grow, on everting the eyelids, opposite the inner margin of the tarsus; in fact, at the point of reflection of the eyelid. It is usual to see a definite row of these tonguelike papillary growths. They resemble the thickened and red synovial fringes seen in tubercular joints. The whole palpebral conjunctival membrane is very red, and there is, as a consequence, slight mucous discharge. This secretion should be carefully examined for Koch's bacillus. It is a curious fact that the pre-auricular gland is not enlarged in these cases. (c) *Lupus*, as a primary affection, is rarely, if ever, seen in the conjunctiva. It is doubtful whether it could be diagnosed in the absence of lupus of the face. Lupus attacks the conjunctiva by spreading over the margin of the eyelid. The conjunctiva becomes nodular and soon ulcerates.

Etiology and pathology.—The cause of these lesions is the bacillus of Koch; its characters are so well known that a description here is unnecessary. It is not easily discovered. The best proof of its presence is the inoculation test, either beneath the skin, or into the anterior chamber of the eye, of a guinea-pig. The discharge or scrapings from doubtful ulcers should always be examined for the bacillus.¹

¹ A coverglass preparation is made; one or two drops of carbol fuchsin are placed upon the slip, which is warmed over a spirit-flame till steam rises. After five minutes the specimen is decolorised in sulphuric acid (1 in 4), washed,

Prognosis and treatment.- From personal observation, I look upon tubercular ulcer of the conjunctiva as a very serious disease. It is a focus, probably primary, of tuberculosis very prone to disseminate and cause death. It is wise, therefore, to destroy the ulcer, either by scraping or excision, and to remove at the same time the large preauricular gland. Tubercular cock's-comb growths should also be freely excised, and every vestige of tubercular-looking tissue taken away so far as this region will allow.

SYPHILITIC AFFECTIONS OF THE CONJUNCTIVA.

Syphilis may attack the conjunctiva in all of its three stages. Chancre of the palpebral conjunctiva, of the cul-de-sac, at the inner canthus, or even on the ocular conjunctiva, may be seen in association with enlargement of the preauricular gland. A conjunctival congestion, or slight catarrh, is a frequent precursor of syphilitic iritis in the secondary stage. As tertiary manifestations, gummata and ulcerations may be met with, the former in the ocular conjunctiva and episcleral tissue. (See Episcleritis.)

The *treatment* is constitutional, and the local use of a mercurial lotion or iodoform powder.

HYPERTROPHY, ATROPHY, AND DEGENERATIVE CONDITIONS OF THE CONJUNCTIVA.

Pterygium is a thickened condition of a part of the ocular conjunctiva. It usually commences opposite to the aperture formed by the opened eyelids, and is more common on the nasal than on the temporal side of the cornea, although it may occupy both these positions in the same eye, or even in both eyes at the same time. Each patch appears in the form of a triangle, of which the apex is directed towards, or encroaches upon, the cornea, the sides being free and formed by a double fold of the mucous membrane, under which a

and counter-stained with Loeffler's alkaline methylene blue for a period of one minute. It is washed again, and mounted, when dry, in Canada balsam dissolved in xylol.

probe can be easily passed. Its colour is generally so similar to that of the conjunctiva that it usually passes unnoticed until it attacks the cornea (see fig. 2, opposite p. 118); sometimes, however, it becomes vascular in structure, and then has a bright red colour. It varies greatly in thickness and in the rapidity of its growth. In some cases it continues for many years without apparent increase; in others, especially those of the vascular kind, the increase may be rapid. In the majority of cases it causes but little or no inconvenience; but when the thickening is great, the conjunctiva is liable to inflammatory attacks. So long as the growth does not extend to the front of the pupillary aperture, the vision is unaffected; but after it has reached this region, the vision decreases in proportion to the extent of the pterygium.

Etiology and pathology.—Pterygium is thought to be caused by persistent exposure of the conjunctiva to irritating substances, and, according to some surgeons, commences as a small abrasion or ulcer opposite the sclero-corneal junction. This theory is absurd; there is no evidence forthcoming in favour of it, and in opposition to this view may be mentioned the rarity of pterygia in this country, in spite of the frequency of marginal ulcers. It is most common amongst persons of middle age and loose conjunctivæ, in those who have travelled or spent some years in hot dusty countries, and in stone-masons and others who are exposed to irritating substances. It may form the starting-point of an epithelioma.

Treatment.—When the cornea is only slightly or not at all involved, and when the increase is evidently slow—that is, where increase is imperceptible during six or twelve months' observation—no treatment is called for.

Where increase is evident, and the pterygium has commenced its march upon the cornea, its removal by operation should be at once resorted to. This can be effected by (1) transplantation, (2) excision, or (3) ligation.

1. *Transplantation* (Desmarres' operation).—The lids being separated by a speculum, the pterygium is seized with forceps and dissected completely away from the cornea and the conjunctiva as far as its base. The lower flap of the incision formed in the ocular conjunctiva by the removal of the pterygium is now enlarged by an

incision of several millimetres in length, made parallel to the lower margin of the cornea. The conjunctiva is then dissected away from the globe to an extent sufficient to receive the pterygium beneath it. The pterygium is then twisted under this flap of conjunctiva and fastened in its new position by one or two fine silk sutures. Finally, the cut edges of the conjunctiva are brought into apposition by similar sutures.

The dissection can be made with 'curved scissors or a Beer's cataract-knife. An excellent little knife is used for this purpose by Anderson Critchett. It is rounded at its extremity, and the cutting



FIG. 29.—Critchett's Pterygium Knife.

edge is continued a short way up the back of the blade. It is made by Weiss. This method gives very satisfactory results; the transplanted conjunctiva soon becomes shrunken and imperceptible.

2. *Excision* is performed in a manner similar to the first stage of transplantation, the mass being cut away at its base by two incisions meeting at the commissure. The edges of the wound are brought together by fine silk sutures. Before inserting the ligatures the ocular conjunctiva should be dissected away from the globe above and below so as to loosen it, and thus enable the upper and lower edges of the wounded surface to be brought into apposition. It is well to unite these close up to the edge of the cornea, otherwise the pterygium is apt to recur.

3. *Ligation* is performed by transfixing the base of the pterygium by several silk ligatures and tying them tightly in such a manner as to involve the whole of the base of the growth, which soon sloughs, and can be removed with forceps.

Pinguecula is a small whitish or yellowish-white tumour of from 1 mm. to 4 mm. diameter, situated in the ocular conjunctiva close to the cornea, and opposite the palpebral fissure. It more commonly occurs on the temporal than on the nasal side of the cornea, but it frequently comes on both sides and in both eyes. It involves the whole thickness of the conjunctiva, with which it moves when the latter is displaced. It is more common after middle age than before that period, also in persons who are exposed by their occupation to irritating vapours and substances. Microscopically, pinguecula is

found not to be composed of fat, as its name implies, but to consist chiefly of condensed cellular tissue; the epithelial layer of the conjunctiva is thickened, and the blood-vessels are obliterated. It causes no trouble or inconvenience; after attaining a certain magnitude it remains stationary. As a rule, no treatment is required, but no harm would be done by its removal.

Amyloid Degeneration of the Conjunctiva.—This is a rare affection, in which there is a soft gelatinous-looking hypertrophy of the conjunctiva, unattended by inflammation or pain. It appears first to attack the sclerotic portion of the conjunctiva, and thence to spread to that of the palpebræ and the fornices. According to Leber, amyloid degeneration is a purely local malady; it may come on as a primary affection of the conjunctiva, or it may be consecutive to chronic granular conjunctivitis. The process consists in the development of amyloid corpuscles or trabeculæ, which are situated in a clear liquid matrix, and are enclosed in a special membrane, containing numerous nuclei. In most instances an amyloid reaction to tincture of iodine and sulphuric acid can be obtained, in some it is limited to the vessels, and in others no reaction at all.

Treatment.—The growth may be excised, or the bulk of the tarsus removed. Scarification or expression may be attempted. The object of treatment is to cure the ptosis caused by the weight of the lid (mechanical ptosis), and to relieve epiphora from eversion of the puncta.

Xerosis (xerophthalmos) is a rare affection, characterised by dryness of the conjunctiva. Dryness may occur from exposure such as is seen in Graves's disease, towards the end of a wasting illness, in facial palsy, and lagophthalmos from other causes, as that due to injury or cicatricial ectropion. There are, however, cases in which dryness occurs without exposure, to which the above term is especially applied. There are two chief varieties, *interstitial* and *epithelial*.

The former is more serious, and involves the whole conjunctival membrane, even the corneal surface. The dryness is accompanied by shrinking, so that in time the retrotarsal folds are obliterated, the movements of the eyeball become limited, and eventually that organ is fixed in the primary

position with the eyelids adherent to it. A narrow palpebral aperture is left. Long before this stage the sight is lost by dryness and superficial vascular changes in the cornea not unlike pannus.

The latter, or epithelial variety, is peculiar in that it is always accompanied with night-blindness (hemeralopia). The dry patches are seen on the ocular conjunctiva opposite the palpebral fissure and either to the inner or outer side of the cornea. The patch is white, dry, and greasy-looking; it has been likened to dry foam. A very serious form may be seen in children (keratomalacia), but in adults it is not a serious affection. Stephenson has recently shown that the visual field for green in these cases is larger than that for red, *i.e.* contrary to the usual colour limitation.

Etiology and pathology.—Interstitial xerosis is the result of some local malady, as trachoma or pemphigus. The change is chiefly in the subepithelial connective tissue, but the epithelium also takes part in this 'dermoid' condition. The epithelium of the cornea, as shown by Treacher Collins, is considerably thickened, and sends down into the altered (cellulo-vascular) substantia propria fingerlike processes resembling the inroads of commencing epithelioma. The superficial cells are flattened, without nuclei, and keratinous.

Epithelial xerosis is limited to the epithelium. The superficial cells undergo fatty degeneration, and on this account the tears do not moisten these areas. An organism has been frequently found in the foamlike desquamation—the xerosis bacillus—first of all by Kuschbert and Neisser, and subsequently by other observers. It has also been discovered, as already stated, in follicular conjunctivitis. It resembles very closely the Klebs-Loeffler bacillus of diphtheria, and, according to Eyre, possesses the following properties: it is an immotile, non-liquefying, facultative anaërobic, non-sporing bacillus. It does not, according to him, show growth on blood serum under thirty-eight hours; if grown in neutral bouillon it does not give an acid reaction, and it does not grow readily on potato. These points serve to distinguish it from the Klebs-Loeffler organism, for, by the microscope, it is difficult. Whether the xerosis bacillus is the actual cause of epithelial

xerosis is not yet proved, and appears doubtful. Uhthoff is opposed to the bacterial view, and believes strongly in alcoholism as a cause.

Treatment is purely palliative. In the interstitial form the local application of glycerine has been advised, but does not give material benefit.

Pemphigus conjunctivæ is a rare affection. It is usually associated with pemphigus of the pharynx, palate, or skin. It is attended with general redness and thickening of the whole conjunctival surface, a gritty sensation as of sand in the eyes, increased lachrymation and photophobia. At first, therefore, it simulates chronic catarrh, but it refuses to yield to ordinary treatment. By careful examination, a small clear vesicle, a greyish discoloured patch, or a slight ulcer may sometimes be found either on the scleral conjunctiva or in the lower cul-de-sac or even on the cornea. The throat, mouth, and skin should be carefully examined for bullæ, as the presence of these would considerably elucidate the diagnosis. The conjunctiva also should be carefully examined day after day, as during the progress of the disease these vesicles present themselves and immediately break down, forming a greyish patch, which is followed by shrinking of the conjunctival tissue. This process may extend over months or even years, but is attended during the whole period by gradual cicatrisation of the whole conjunctiva, which finally becomes whitish, cloudy, dry, smooth, and tense. First the upper and lower culs-de-sac disappear, then there are cicatricial bands extending between the lids and the globe, until these become quite adherent to each other. The cornea at the same time becomes cloudy and dry, and the vision is greatly deteriorated, or entirely lost. The prognosis is very unfavourable, especially as the disease usually attacks both eyes, either at the same time or in succession.

Many cases of shrinking of the conjunctiva have occurred in which no trace of bullæ of the conjunctiva has been observed; so much so, that von Graefe described them as *essential shrinking*. In the light, however, that has been thrown upon the subject by Steffan, Bäumler, and Lang, it is possible that these may have been overlooked, and that most, if not all, cases of this class are due to pemphigus.

Treatment is unavailing to check the progress of the disease. Moist protection with vaseline or glycerine, especially during the night, seems to comfort the patient. Arsenic has been recommended internally, but nothing is satisfactory.

TUMOURS OF THE CONJUNCTIVA.

Tumours of the conjunctiva are *benign* and *malignant*.

The **innocent growths** include osteoma, lipoma, papilloma, dermoid fibroma, angioma, and cysts. (For granuloma see **Exposed sclera.**)

Osteoma and *lipoma* are found in the upper and outer part of the conjunctival sac, and are seen projecting downwards when the eyelid is everted. The bony growth is always covered by a thin layer of conjunctiva. It is probably congenital in origin, with a tendency to increase in size about puberty. Hartridge¹ records a good example, and refers to several others. Lipomata are really beneath the conjunctiva, and are easily shelled out after incising that membrane. A *papilloma* may grow at the limbus or from the plica semi-lunaris, or even on the inner lip of a slit-up canaliculus. At the sclero-corneal junction it has a tendency to invade the cornea, so early removal is advised. A *dermoid fibroma* (*conjunctival mole*, Bland Sutton) must be distinguished from a dermoid cyst. The conjunctival dermoid is solid, composed chiefly of dense fibrous tissue, a few glandular elements, some fine hairs, and is found more often than not at the sclero-corneal junction opposite the palpebral fissure, involving both conjunctiva and cornea. It may, however, be seen away from the cornea, occupying the ocular conjunctiva exposed by a cleft in the eyelid, or even on the caruncle. Trichosis bulbi has been applied to small dermoids in which the hairy element is predominant. In the absence of hairs and glandular structures the dermoid has been called a fibroma. It is, however, in the position of a dermoid, is congenital, usually small, pearly white, and of firm consistence. It does not recur after removal, though the cornea retains a white blemish where the tumour had encroached upon it. A *nævus* limited

¹ *Ophth. Soc. Trans.* vol. xv. p. 51.

to the conjunctiva is very rare, but one spreading from the eyelid on to the conjunctiva at the inner canthus is not uncommon, and causes the caruncle and plica to be turgid and florid red. Cavernous nævi are raised, almost black, and increase in volume on any exertion. According to Snell, who has published three cases of primary angioma of the conjunctiva, a cavernous growth has a striking resemblance to a blackberry. A nævoid condition of the ocular conjunctiva to the inner and more especially to the outer side of the cornea is frequently developed in alcoholic individuals.

Cysts are of two chief kinds—the simple serous cyst and the parasitic, or *cysticercus cellulosæ*. The *serous cyst*, like a small vesicle, is often seen on the ocular conjunctiva; a prick with a needle and it disappears. It is probably a dilated lymphatic space or vessel. It rarely exceeds the size of a pea. The *cysticercus* of the *tænia solium* is very rare in this country. It is much larger than the simple cyst, and often has an opaque wall from the thickening of the subconjunctival tissue around it. If translucent, the head of the parasite can be recognised as an opaque white spot.

Treatment.—With the exception of nævi, there is but little difficulty in dealing with these tumours or cysts. If a dermoid is present at the sclero-corneal junction it should be shaved off with a Beer's cataract-knife flush with the corneal surface, and the conjunctiva made free and brought together with sutures close to the corneal margin. Venous nævi should not be destroyed by the injection of coagulants, but either removed with scissors or destroyed by electrolysis or ethylate of sodium. Snell used the latter with success in two instances (see p. 16).

Malignant tumours consist in epithelioma and sarcoma, both of which attack the conjunctiva at the limbus. Sarcoma may occur elsewhere—for instance, on the plica or caruncle—but epithelioma invariably develops in this situation. Involvement of the conjunctiva secondarily by rodent ulcer is not uncommon. Similarly, growths may invade it from behind.

Epithelioma is an opaque white tumour with a surface of a granular or cauliflower appearance. It is more an outgrowth than an invader of deep tissues, and will spread over the

whole cornea and greater part of the ocular conjunctiva before entering the eyeball. Around the growth are seen large varicose blood-vessels feeding it. It appears after middle age, recurs after each removal, may bleed or ulcerate, and will eventually prove fatal by dissemination. Cell-nests are sparingly scattered throughout the dense masses of epithelium which surround the branched papillary processes of vascular connective tissue. Cell-inclusion is seen, as well as irregular mitotic figures (see p. 19).

Sarcoma appears as a gelatinous, semi-translucent growth with pigmented spots or nodules, at the sclero-corneal junction. Like epithelioma, it occurs late in life, does not easily penetrate the eyeball, and has, coursing towards it, large tortuous blood-vessels. It is almost invariably pigmented—indeed, sometimes quite black—and it has a smooth uniform surface. Besides these two points, it is unlike epithelioma in that there is no tendency for it to ulcerate; nor does it attack the cornea, but overlaps it, so that a probe may be passed beneath. It is doubtful whether these melanomata are always sarcomatous; their intimate structure requires further elucidation.

Treatment.—Removal of the tumour from the eyeball is not successful; excision of the eye, even if the vision is good, be it sarcoma or epithelioma, is the best procedure, and, along with it, all the conjunctiva, unless the growth is very limited in extent.

CHAPTER IV.

THE CORNEA.

ANATOMY AND PHYSIOLOGY—CLINICAL EXAMINATION—INTERSTITIAL KERATITIS—VASCULAR KERATITIS, OR PANNUS—SUPERFICIAL OR ULCERATIVE KERATITIS—PHLYCTENULAR KERATITIS—HERPES CORNEÆ—DENDRITIC KERATITIS—FILAMENTARY KERATITIS—BULLOUS KERATITIS—RODENT ULCER—SUPPURATIVE KERATITIS—NEUROPARALYTIC KERATITIS—KERATITIS E LAGOPHTHALMO—SEQUELÆ OF CORNEAL ULCER—ENUCLEATION AND ITS SUBSTITUTES—DEGENERATIVE CONDITIONS—CONGENITAL CONDITIONS—CONICAL CORNEA—TUMOURS—DEPOSITS IN CORNEAL TISSUE.

ANATOMY AND PHYSIOLOGY.

THE human eyeball is suspended in the front part of the orbit by various muscular and fascial attachments; not the least in importance is the suspensory ligament of Lockwood. Behind, and somewhat to the inner side, the optic nerve enters it like a stalk. With the exception of its anterior surface, the globe is opaque and white. Surrounding it is a serous sac, the capsule of Tenon, with a visceral and parietal layer lined by endothelium, analogous to the peritoneal or pleural cavities, which serves as a ball-and-socket joint for the free movement of the eyeball, and is pierced by tendons, vessels, and nerves.

The eyeball is almost spherical, and, roughly speaking, has a diameter of one inch. It is composed of three tunics: an outer *fibrous*—the cornea and sclera; a middle *vascular*—the iris, ciliary body, and choroid; and an inner *nervous*—the retina. The transparent contents—the aqueous humour, the crystalline lens, and the vitreous body—together with the cornea, constitute the refracting media of the eye.

The *cornea*, or transparent anterior portion of the tunica fibrosa, projects forwards beyond the contour of the sclera, owing to its radius of curvature being considerably less (7·7 mm.). It fits into the anterior opening of the sclera like a watch-glass, the posterior surface of which is bevelled at the expense of the anterior. This

beveling is usually greater in the vertical diameter; consequently, as the posterior surface of the cornea is circular, the anterior is oval in appearance, having a transverse diameter of 12 mm. compared with 11 mm. in the vertical. The corneal arc occupies about one-sixth the circumference of the eyeball. It is not an arc of a circle, but of an ellipsoidal body; as a consequence, the curvature varies in different meridians, the radius of curvature in the vertical being usually less than that in the horizontal. It will later be shown that astigmatism is mainly corneal. Its thickness is greater at the periphery (1.1 mm.) than at its centre (0.9 mm.). Acting as a lens by itself, therefore, it would be a diverging meniscus.

In the cornea we find from before backwards five layers—the anterior epithelium, Bowman's membrane, the substantia propria, the posterior elastic lamina, or Descemet's membrane, and the posterior epithelium.

The *anterior epithelium* is of the stratified variety, arranged in several layers, the deepest of which is composed of columnar cells with oval nuclei: this layer regenerates the epithelium if abraded or destroyed by ulceration; then follow two or three layers of polyhedral cells with spherical nuclei; lastly, there are two or three layers of flattened cells with discoid nuclei. The polyhedral cells are separated from each other by intercellular spaces, which are bridged across by numerous processes, the cells being known as prickle-cells. Through these spaces the fluid for the nutrition of the cells passes. This epithelium is continuous with that of the conjunctiva, from which it differs in being somewhat thicker.

Bowman's membrane is the transparent homogeneous anterior part of the substantia propria. It was considered by Bowman to be elastic; but it is stained pink with picro-carmin, and splits up into fibrillæ like the substantia propria with permanganate of potash, unlike elastic tissue. It is firmly adherent to the subjacent tissue; but if stripped off, it rolls up like elastic tissue. If destroyed by injury or inflammation, it is not regenerated. It differs from elastic tissue in being soluble in mineral acids and in boiling water.

The *substantia propria* is continuous with the sclerotic; it consists of numerous parallel lamellæ of modified fibrous tissue. Each lamella is composed of closely woven bundles of white fibrils saturated with a chondrin-yielding cement substance which imparts to this ordinary fibrous tissue its transparency. The lamellæ are numerous, sixty or more in number, and are intimately blended together by obliquely set bundles of fibrils as well as by the cement substance. Between the lamellæ are found lacunar spaces intimately

connected with one another in the planes and through the bundles by a system of anastomosing canaliculi, constituting the lymph canalicular system of Recklinghausen. These anastomose freely with the lymphatics of the conjunctiva at the circumference of the cornea. They must not be mistaken for Bowman's tubes, which are a system of straight lines of cleavage produced artificially by the injection of mercury. Each bundle of fibres possesses a fusiform nucleus—the fixed cells of the cornea. It was formerly supposed that the lymph lacunæ contained branched corneal corpuscles analogous to osteoblasts in osteal lacunæ; this, however, is now doubted by some histologists, especially by Hamilton,¹ whose views appear convincing. Besides the fixed cells of the cornea, there are a few migratory cells which enter from the periphery and travel along the lymph-channels. In injury or inflammation the entry of migratory cells is enormous. The cornea is nourished by the circulation of plasma through its lymphatic system.

The *posterior elastic lamina*, or Descemet's membrane, is strongly resistant, possesses elastic properties, stains orange-red with picrocarmine, and dark purple with logwood, from which facts it is not typical elastic tissue. It is, however, very resistant to hot water and the mineral acids. At the circumference of the cornea it becomes split up into a leash of fibres to form the *ligamentum pectinatum*: these pass to the iris and ciliary body; they also give attachment to some fibres of the ciliary muscle and dilator pupillæ. Its posterior surface is lined by a single layer of flattened nucleated cells. These cells are continued along the fibres of the *ligamentum pectinatum*, and over the anterior surface of the iris.

The *nerves of the cornea* are derived from the ciliary nerves; they enter the fore part of the sclerotic, and thence pass to the *substantia propria* of the cornea. They retain their dark outline for about 1 mm., but then become transparent, and form a plexus throughout the laminated structure. From this primary plexus other nerves proceed to form a finer plexus just beneath the epithelial layer, and this gives off branches between the epithelial cells, to form a still more superficial network. Around the larger nerves are lymphatic spaces.

All round the periphery of the cornea is a *fringe of capillaries*, branches of the anterior ciliary vessels; these are continuous with the vessels of the *limbus conjunctivæ*, and extend for about 1.5 mm. into the corneal tissue; beyond this the cornea is absolutely devoid of blood-vessels in its normal condition.

¹ *Textbook of Pathology*, vol. i.

Lymphatic system of the cornea.—The lymph in the corneal lacunæ arises from three sources: (1) from the capillary loops at the corneal circumference; (2) from the anterior chamber by way of the spaces of Fontana, the canal of Schlemm, and the sclerotic lymphatics; (3) from the anterior chamber by diffusion through the posterior corneal endothelium, and the membrane of Descemet. The amount of lymph derived by this last path during life is probably quite small. The lymph passes away chiefly by the conjunctival lymphatics, though to a slight extent by means of the perineural lymph-spaces.

Development of the cornea.—The anterior epithelium is developed from ectoderm, the rest of the cornea being mesodermic. After the budding off of a portion of the ectoderm to form the lens, processes of mesoblastic tissue grow in between the superficial ectoderm and the primitive lens. This tissue splits into two, the anterior portion forming the substantia propria, membrane of Descemet and posterior epithelium, of the cornea, the posterior finally producing the vascular pupillary membrane. The space between is the anterior chamber.

CLINICAL EXAMINATION.

The cornea may be examined in various ways - by inspection, and, more carefully, by focal illumination, with the ophthalmoscope, and by testing its sensibility. The normal cornea is transparent, is highly sensitive, is devoid of blood-vessels, and has a smooth, uniformly convex surface. Any opacity, vascularity, irregularity, or altered sensibility points to some present or past morbid change. The senile degeneration, or arcus senilis, is an opacity which can hardly be classed in the category of disease. It interferes in no way with the healing of a corneal incision.

Oblique focal illumination is a useful method of examining the cornea, also the anterior chamber, the iris, the crystalline lens, and the anterior part of the vitreous. It should be adopted as a routine practice in all cases where the presence of a foreign body or of disease in these parts is suspected. The patient should be seated in a dark room, with the ophthalmoscope lamp placed about 30 cm. to 40 cm. to the temporal side and slightly in front of the plane of the patient's face. Then by using a convex lens of 14 D or 16 D, and slightly changing the position of the lamp and lens, the light can be

easily brought to a focus upon either of these structures. The part thus illuminated can at the same time be magnified by using a second lens held in the other hand (see fig. 30). This lens should be held at its own focal distance from the part to be viewed: the rays are then parallel as they reach the observer's eye, E' .

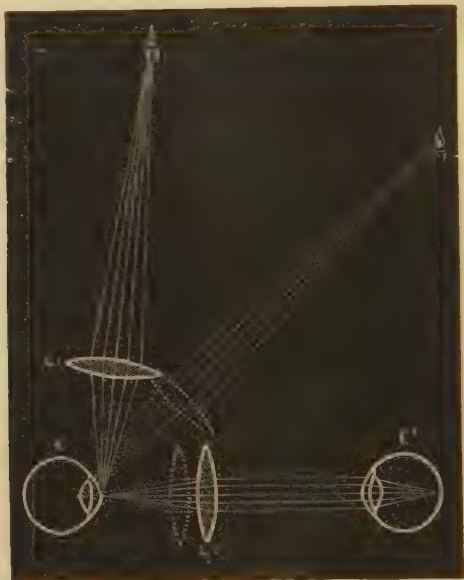


FIG. 30.—Oblique Focal Illumination.

E , patient's eye; E' , observer's eye; L_1 , condensing lens;
 L_2 , magnifying lens.

By inspection and focal illumination it is necessary to note the size, shape, and curvature of the cornea, the presence of irregularities of its surface, superficial or deep opacities, superficial or deep-seated vessels, and differentiate between lesions in the cornea and morbid deposits in the anterior chamber. With the ophthalmoscope, by the direct examination atrophied vessels may be seen in the cornea with a strong convex lens (+20 D), and by retinoscopy the curvature of the cornea may be noted. Its sensibility may be tested with a piece of soft rolled-up blotting-paper.

KERATITIS.

Keratitis (corneitis), or inflammation of the cornea, occurs in various forms and degrees of severity. As the cornea may be divided anatomically into three parts—*conjunctival* portion, or the anterior epithelium and Bowman's membrane; *scleral* portion, or the substantia propria; and the *uveal* portion, or Descemet's membrane and the posterior epithelium—so inflammatory changes may be classed into superficial, interstitial, and deep. All forms of keratitis are accompanied by more or less circumcorneal redness (its absence excludes corneal inflammation), loss of transparency, and such functional disturbances as ciliary pain, photophobia, lachrymation, and impairment of vision; and in many may be found newly formed blood-vessels, and irregularity of surface. The surrounding redness is due to the injection of the conjunctival or episcleral vessels, or both, and the cloudiness to infiltration of the cornea with leucocytes which enter from the periphery. As the ocular conjunctiva is loosely connected with the subjacent sclera, it is easy to move this membrane, and so distinguish between superficial and deep-seated redness; the latter, moreover, is not bright red, but has a violet hue.

1. **Interstitial keratitis** (syphilitic, parenchymatous) is a diffuse inflammation of the whole substantia propria of the cornea.

Clinical features.—In this form of keratitis, the whole cornea undergoes a chronic inflammatory change, and evinces no tendency either to the formation of pus or to ulceration. First, there is slight congestion of the vessels in the ciliary region around the margin of the cornea, then a diffused greyish opacity at the centre; this may be so slight that it is recognised only by focal illumination; soon, however, it becomes decidedly cloudy, some of the opacity being near the surfaces, and other patches deeper. This cloudiness, or ground-glass appearance, gradually extends over the whole cornea, until the pupil and iris are more or less hidden from view. In the most severe cases the opacity assumes a yellowish tint, and no trace of the iris can then be seen, even with the oblique illumination. The degree of pain, photophobia, and

congestion of the conjunctiva is variable. In some cases these symptoms are very slight from the beginning to the end of the case; in others the eyes are extremely hypersensitive to light, very painful, with profuse lachrymation, and the ocular conjunctiva is much congested.

In most cases, very minute blood-vessels are formed in the layers of the cornea. These are derived from the episcleral branches of the anterior ciliary vessels; they are extremely fine, and their separate branches can be distinguished only by means of a magnifying lens, when they appear in the form of a fine network of branches which are given off from a larger trunk at the periphery of the cornea. These vascular areas are not of a bright red colour, unless they are very near the surface; when deep down in the corneal tissue their colour is modified by the opacity, and they appear to be of a dull reddish colour—the ‘salmon-patch’ of Hutchinson. They may occur in any position; and often attack the upper or lower margin of the cornea. Fig. 4, opposite p. 118, represents a severe case of this disease. The whole cornea is opaque, and a salmon-patch is seen over its upper third.

In some forms of parenchymatous keratitis the opacity is most dense between the centre and periphery, like a greyish-yellow ring. In others, especially those due to acquired syphilis (see Etiology), the opacities are numerous, deep-seated, and discrete.

Diagnosis.—It is sometimes difficult to diagnose between superficial forms of keratitis and those which attack chiefly the substantia propria. In *superficial keratitis* the corneal surface is usually rough and uneven. When vessels are present, they may be traced directly from the cornea across the limbus on to the conjunctiva; they are clearly defined, having a bright red colour, and they branch off in an arching manner like the branches of a tree. In *interstitial keratitis* the surface of the cornea may be lustreless, but it is not rough or uneven. When vessels are present, they cannot be traced from the ocular conjunctiva across the limbus, but they disappear beneath the latter in order to reach the sclera. They are not clearly defined, but require a magnifying lens to render them distinct; they are of a dull ‘salmon’-red colour,

and their branches are given off in a nearly parallel (birch-like) way.

Complications are not unfrequent in the tissues of the neighbouring parts. The most common of these are iritis and cyclitis. Diffuse keratitis usually attacks both eyes, but as a rule one cornea is first invaded and rendered fairly opaque before the attack in the second eye commences. The interval between the attacks in the two eyes varies from two or three weeks to as many months. It generally occurs between the ages of six and fifteen; although it is sometimes seen as early as three, and has been known as late as thirty-five years.

The *duration* of this affection under proper treatment is on an average from about six months to a year; but severe cases are two years or longer before they become stationary.

Vision is nearly always somewhat impaired after this disease. The cornea may look very transparent, and only the faintest haze may be detectible by focal illumination; but this will almost invariably be found to interfere with distinct vision.

Etiology and pathology.—Interstitial keratitis is a chronic inflammation of the whole cornea, especially of its deeper layers. It is brought about by constitutional diseases—syphilis and tubercle. It is now almost universally admitted that the inflammation does not start primarily in the cornea, but extends to it from the circumcorneal zone. If the surrounding conjunctiva and subjacent sclera are the chief seat of the inflammation, the superficial layers of the substantia propria will be most involved and the vessels invading the cornea visible with the naked eye; whereas if the inflammation is more deeply seated, as is the rule, in the uveal tissue and adjacent sclera, the deeper layers of the cornea will be more infiltrated and the individual vessels invisible without the aid of a lens. It happens in the worst cases that the cornea is vascularised throughout its whole substance.

The cause, *par excellence*, of interstitial keratitis is hereditary syphilis, a fact brought prominently forward by Hutchinson in 1863. It is only necessary to note the frequency of collateral evidences of congenital syphilis in these cases to dispel any opposite opinion. At least 70 per cent. may be attributed to this cause. Contrary to what would be

expected, the poison of acquired syphilis seldom attacks the cornea; so rare is it, that very few instances have been placed on record. A form in acquired syphilis, called by Mauthner *keratitis punctata syphilitica*, is described by him as consisting of punctiform infiltrations of the deeper layers of the *substantia propria*. It is identical with the *keratitis interstitialis punctiformis specifica* of Hock. Greeff states that syphilis acquired in infancy is more prone to be followed by interstitial keratitis. This is of interest in that it points to the cornea in childhood—*i.e.* during its growth—being more susceptible to the syphilitic virus. Apart from syphilis, tuberculosis as a cause is strongly upheld by von Hippel and other observers.

Pathological anatomy shows the cornea to be considerably thickened, softened, and densely infiltrated with leucocytes. Here and there are seen giant cells, and, on its posterior surface, collections of round cells, some containing pigment-granules (*descemetitis*). The ciliary body and iris are found infiltrated with round cells, with other evidences of inflammation. The surrounding zone of sclera is, like the cornea, thickened, and its fibrillar structure hidden to view owing to the dense masses of leucocytes. Blood-vessels in great numbers are seen in the sclera and cornea, giving to these membranes the appearance of granulation tissue. Throughout life these vessels are visible in the cornea as fine threads, their atrophied remains bearing testimony of the former *keratitis* (fig. 31).

The recognition of the cause depends to a great extent upon the collateral signs of hereditary syphilis. The patient presents a very peculiar physiognomy, of which a coarse flabby skin with *café au lait* complexion, pits and scars on the face and forehead, the definite outline between the latter and the scalp being destroyed by irregular short downy hairs, cicatrices of old fissures at the angles of the mouth, a sunken bridge to the nose, deafness, and a set of permanent teeth peculiar for their smallness, bad colour, and the semilunar notch of the upper central incisors, are the most striking characters.¹

¹ *Syphilitic Diseases of the Eye and Ear*, by Jonathan Hutchinson, p. 30.

Other symptoms of inherited specific disease can often be detected in the brothers and sisters of the patient, and the history of acquired syphilis can often be elicited from one of the parents, either directly from their own statements, or indirectly by interrogation. Thus it will frequently be found that the mother of the patient suffered from numerous miscarriages, or that several children prior to the patient were either prematurely born, still-born, or died in early infancy, often with specific symptoms.

Treatment must be directed to the improvement and support of the general health as well as to the local condition. The subjects of this disease are generally weak, and frequently anæmic. It is important that they should be placed under the best hygienic conditions, that they should have abundance of nutritious food, and plenty of exercise in the open air. Strong alcoholic drinks should be scrupulously avoided. A prolonged course of mercury should be prescribed in syphilitic cases; any of the mercurial preparations will answer the purpose, such as pil. hydrargyri, hydrarg. cum cretâ, the perchloride, &c., or the inunction of unguentum hydrargyri in the inner aspect of the arms and thighs, where the skin is soft and devoid of hair. Whichever form is prescribed, it should be continued for a long time; but its action must be carefully watched lest salivation be produced. The state of the gums and inside of the lips should be examined at each visit; and any sponginess being observed, the medicine should be stopped until these symptoms have disappeared. Subconjunctival injections of cyanide of mercury have been used. For an account of this method of treatment see p. 135. In addition to mercury, the internal administration of cod-liver oil, of the syrup of phosphates of iron, quinine, and strychnine, of the syrup of the iodide of iron, or of the perchloride of iron with quassia, is a very valuable adjunct. The eyes should be shaded from bright light and exposure to cold by means of tinted glasses; if there is photophobia, they had better be closed, and covered



FIG. 31. — Atrophied Corneal Blood-vessels.

by small pads of cotton-wool and a bandage. A $\frac{1}{2}$ per cent. solution of atropine should be systematically dropped into the palpebral aperture once or twice daily throughout the active, inflammatory stage: this not only acts as a sedative, but prevents iritis; if photophobia be very acute, cocaine may be added. When the inflammation is subsiding, and there is no photophobia, the use of the yellow oxide of mercury ointment (F. 39) is advisable, as it promotes absorption of the opacity; it should be put into the palpebral aperture, and massage applied through the lids, night and morning.

2. **Vascular keratitis, or pannus**, is a superficial vascularity and opacity of the cornea (fig. 1, opposite p. 118).

Pannus is seen most frequently in the upper half of the cornea. The upper eyelid is slightly drooped, and upon raising it the cornea appears steamy or cloudy, and numerous tortuous vessels are traceable from the ocular conjunctiva on to, as it were, the corneal surface. They are visible as vessels to the naked eye, but their beautiful arborescent appearance is seen to advantage by means of a lens and focal illumination. The lower part of the cornea may be also slightly cloudy and vascular, leaving only that portion just below the horizontal meridian transparent; or the whole cornea may be covered with vessels and uniformly hazy. There is slight photophobia, lachrymation, and more or less muco-purulent discharge. These vessels are continuous with those of the limbus conjunctivæ, and, when sufficiently far apart to be seen separately, present a tortuosity which distinguishes them from those met with in other forms of inflammation of the cornea. Their number varies with the severity of the case; sometimes they are only three or four in number, and the opacity so slight as to be hardly perceptible. In the severer forms they are so numerous as to constitute a thick fleshy-looking web, and the opacity of the subepithelial tissue is so great that the patient's vision is reduced to mere perception of light. In such a case the cellular deposit invades and destroys the substantia propria, the deeper laminae only being seen in their natural state upon microscopic examination. In this cellular new-growth lymphoid follicles similar to those present in the fornix conjunctivæ may be found.

Etiology and pathology.—Pannus is caused in most

instances, directly or indirectly, by granular conjunctivitis: directly, by the irritation of the rough granular lid as it rubs upon the cornea; and indirectly, by the edge of the incurved tarsus, the sequel of trachoma. Pannus may also be caused by trichiasis and distichiasis, the sequel of blepharitis. Knies, however, believes that trachomatous tissue is actually developed in the superficial layers of the cornea; and it seems probable that, in the most severe forms of the disease, such is the case. Examined microscopically, there is found between the superficial epithelium and Bowman's membrane a thin layer of granulation tissue, *i.e.* an infiltration of round nucleated cells permeated with blood-vessels which have grown in from the limbus conjunctivæ.

Prognosis.—If the cause of the keratitis can be successfully dealt with, the disease will soon disappear, often in a few weeks. As the causes—especially entropion—are frequently difficult to cure, pannus may last many weeks or months, varying in severity—one week much better, the next week as bad, or worse than ever. If complicated with ulceration there will be some permanent opacity; whereas pannus by itself may disappear entirely, without any deterioration in visual acuity.

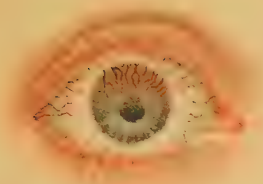
Treatment.—In all cases of pannus we have to direct our attention to the removal of two conditions—*viz.* the granular condition of the conjunctiva and the vascular web and opacity of the surface of the cornea. As the morbid condition of the cornea is secondary to granular lids, that should first be treated (see Granular Conjunctivitis); and it will almost invariably be found that the cure of the granular roughness of the lids and of any trichiasis or entropion that may be causing friction of the cornea will be followed by a gradual clearing-up of the cornea. It may be many weeks or months before satisfactory transparency is established, but the improvement will, nevertheless, be very marked.

The operation of *peritomy* (syndectomy) is sometimes performed in cases of pannus. It consists in the removal of a zone of conjunctiva about 4 mm. in width, and episcleral tissue, from the immediate vicinity of the cornea. New fibrous tissue is formed, which subsequently contracts, and strangles any new vessels which may have developed.

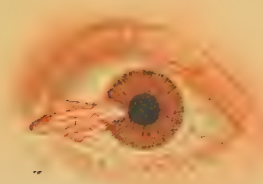
DESCRIPTION OF PLATE.

- FIG. 1.—Pannus (partial).
„ 2.—Pterygium.
„ 3.—Local Keratitis.
„ 4.—Interstitial Keratitis (Salmon-patch above).
„ 5.—Punctate Keratitis.
„ 6.—Ulcer of Cornea (healing).
„ 7.—Phlyctenular Conjunctivitis.
„ 8.—Plastic Iritis.
„ 9.—Gummatous Iritis.
„ 10.—Posterior Synechiæ (Atropine has been used).
„ 11.—Hypopyon.
„ 12.—Hyphæma.

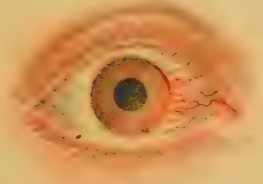
Plate I.



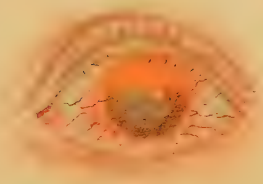
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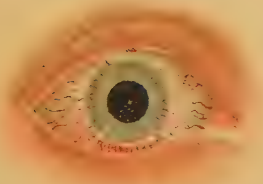
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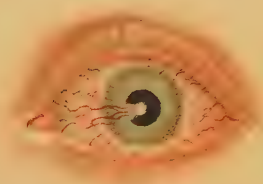
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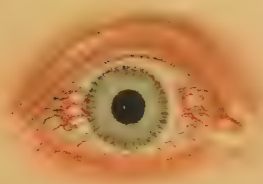
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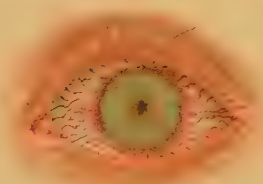
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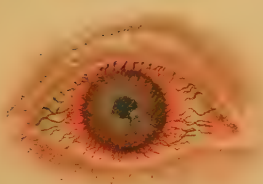
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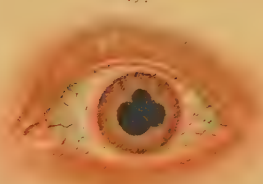
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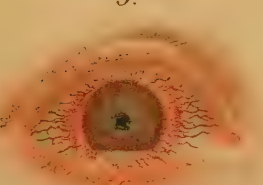
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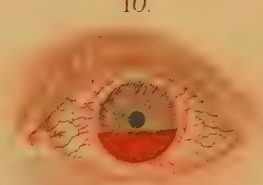
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10.



11.



12.



When pannus has been allowed to become complete, so that a fleshy-looking vascular web has formed over the whole cornea, and no transparent portion remains, *inoculation of pus* is sometimes performed. The process consists in simply transferring some purulent matter from the eye of an infant during the first week of an attack of ophthalmia neonatorum into the palpebral aperture of the patient. An acute attack of purulent conjunctivitis is thus established, and is sometimes followed by clearing up of the cornea. Not unfrequently, however, the process is followed by complete destruction of the eye. The contagious and destructive nature of this remedy renders it very objectionable. It should only be adopted as a last resource.

The artificial production of purulent conjunctivitis by means of jequirity, as a means of cure both for granular lids and for pannus, is a milder remedy for this extreme condition, but is still fraught with considerable danger of causing panophthalmitis. It should only be employed in chronic persistent cases where other remedies have failed, after patient trial, and in which there is considerable opacity of the cornea. It is particularly dangerous where ulceration of the corneal surface is present. An infusion of the seeds of jequirity¹ is used for this purpose. It is prepared as follows: Take 3 grammes of the pulverised seeds, and macerate for twenty-four hours in 500 grammes of cold water, and then add 500 grammes of boiling water. Allow the infusion to cool, then filter immediately.

This infusion is introduced into the palpebral sac three times in the day. If the resulting irritation is severe, this will be sufficient, otherwise the application must be continued on the second, and, if necessary, on the third day. It is followed in a few hours by severe irritation of the ocular and palpebral conjunctiva. Acute inflammation follows the next day; the patient can no longer open his eyes; the lids are cedematous, and there is serous secretion, which is sufficiently copious to drop from the lids if the patient lowers his head. This continues for several days, and is accompanied by pyrexia, sleeplessness, headache, and constipation. After the third day the period of suppuration sets in, and lasts about five days.

¹ *Annales d'Oculistique*, August 1882, p. 24. Also see *Ophthalmic Review*, vol. ii. p. 19.

The suppuration then gradually decreases, and the patient begins to feel improvement up to the fifteenth day, about which time he is finally free from inflammation, and is often cured of his granulations, whilst the cornea gradually begins to clear.

3. **Superficial or ulcerative keratitis** is an inflammation of the cornea beginning in the corneal epithelium. Consequently it includes all the varieties of corneal ulceration, some of which, however, may also involve the deeper layers of the cornea. It constitutes an important part of ophthalmic practice, especially among the children of the poor.

General features.—All forms of superficial keratitis give rise to severe symptoms—lachrymation, photophobia, and blepharospasm. There is always circumcorneal redness. A local cloudiness of the cornea from infiltration, or a phlyctenula or vesicle, is followed by a breaking down of the epithelium and superficial lamellæ, and molecular loss of tissue occurs, which constitutes ulceration. The amount of destruction varies considerably in depth and extent. The cornea presents an irregular surface; and if a drop of fluorescein is instilled, the ulcerated area will stain green.

The varieties of ulcerative keratitis are as follows :
i. Simple superficial ulcer of the cornea ; ii. Phlyctenular keratitis ; iii. Creeping phlyctenular ulcer ; iv. Herpes corneæ ; v. Dendritic keratitis ; vi. Filamentary keratitis ; vii. Bullous keratitis ; viii. Rodent ulcer ; ix. Suppurative keratitis, including (a) Deep ulcer, (b) Serpiginous ulcer, and (c) Corneal abscess ; x. Neuroparalytic keratitis ; xi. Keratitis e lagophthalmo ; and xii. Secondary ulcerative keratitis.

i. **Simple superficial ulcers** of the cornea are usually circumscribed, and often so transparent that they may escape attention unless oblique focal illumination is used. By this method of examination, however, they can always be detected, and are usually found to have margins more or less infiltrated and opalescent. Any doubt as to the existence of an abraded surface or ulcer of the cornea can be at once dispelled by the use of solution of fluorescein (F. 18), a drop of which, when placed in the palpebral sac, will at once colour the denuded portion bright green. Both ulcers and abrasions are attended

by severe photophobia, lachrymation, and neuralgic pains in and around the eye. When situated near the centre of the cornea, vision is much interfered with ; when peripheral, it is but slightly affected.

These ulcers are often of traumatic origin, being caused by a slight scratch or blow, or by the presence of a foreign body. They are more liable to occur in those who are debilitated either from senility, alcoholic excess, or disease. They sometimes come on during the course of an attack of conjunctivitis, more especially in the phlyctenular form. As a rule, early and proper treatment will cause healing of the ulcer without leaving any permanent opacity.

The *treatment* is similar to that of phlyctenular keratitis (which see).

ii. Phlyctenular keratitis (pustular keratitis) is characterised by the appearance of one or more pustules on the surface of the cornea. They are similar to those occurring in phlyctenular conjunctivitis (p. 93) ; in fact, both cornea and conjunctiva are frequently attacked together. Each consists of an aggregation of cells immediately beneath the epithelial layer. They occur simultaneously or in successive crops. They attack any part of the cornea, but are frequently found near the sclero-corneal junction. At the end of three or four days they rupture, and form one or more superficial ulcers. A phlyctenular ulcer is, as a rule, a simple ulcer—*i.e.* it readily yields to mild remedies ; it is usually small, circumscribed, and does not spread. It may, however, become infected, and either take on serpyiginous action, spreading superficially, or bore through the thickness of the cornea.

Photophobia and lachrymation are marked symptoms ; the former is so great as to cause acute blepharospasm. The ocular conjunctiva is usually injected, and may contain similar pustules. As the ulcer heals, a leash of vessels is developed between it and the margin of the cornea.

This affection is common in strumous children, and occurs more frequently amongst the poor and ill-fed than the well-to-do. An exaggerated condition of this disease is, indeed, called *strumous keratitis*. It differs from the phlyctenular form in degree rather than in kind. The ulcers are

preceded by a cluster of phlyctenular bodies, which disintegrate and form extensive areas of ulceration. They are multiple, usually symmetrical, eccentric or marginal at first, but by their size often reach over the centre of the cornea. They become vascular, are prone to suppurate, are liable to perforate, and frequently recur. In short, these ulcers are the most troublesome of all, and, even if cured and recurrence prevented, they leave such extensive opacities and irregular astigmatism that sight is severely and permanently impaired. The symptoms are proportionate. The lachrymation is profuse; the scalding tears spurt out when any attempt is made to separate the tightly clenched eyelids. The eyelids are swollen, red, and streaked with distended and tortuous veins; the cheeks and upper lip are frequently eczematous and swollen. A muco-purulent rhinitis is not an uncommon accompaniment.

A variety of phlyctenular keratitis, and one frequently overlooked, presents distinctive features: numerous small, white, vesicular-looking spots occur at the sclero-corneal margin, accompanied by intense photophobia and lachrymation, marked miosis, and considerable conjunctival injection. Slight chemosis of the conjunctiva often hides the trouble, and the condition is put down as one of catarrhal ophthalmia. The contraction of the pupil, the intensity of the symptoms, together with the absence of lymph-flakes in the culs-de-sac, readily clear up the doubt. The disease is usually symmetrical, and liable to recur.

Treatment.—Improvement of the general health is a cardinal point in the treatment of all forms of superficial keratitis. The patient should be placed under the best possible hygienic conditions: good food, plentiful exercise in the open air, and the internal use of tonic medicines, such as iron, quinine, ammonia and bark, or strychnine, as the nature of the case may indicate.

Friction of the lids against the ulcer by constant blinking and exposure to lid must be prevented by means of a light pad and bandage. Should, however, there be much discharge, it must be allowed free exit, and, indeed, be irrigated with weak antiseptic lotion, a large black or green shade over

both eyes replacing the single pad ; or neutral-tinted goggles with gauze sides are often better for children, as the child is less likely to bow the head.

The following applications should be used locally. In the early stages, while the symptoms are severe, sedatives should be adopted ; and in my experience nothing acts better than the use of atropine (F. 10). It should be instilled three times a day. Compresses of hot or cold boracic acid lotion (F. 28) will often relieve the burning pain in the eyes. The tears are apt to cause eczema on the eyelids and cheek, which can be prevented by smearing some pure vaseline over the part.

Later, when the symptoms are subsiding, and the child is able to open the eyes without much discomfort, yellow oxide of mercury ointment (F. 39) alone, or combined with atropine and cocaine, will act as a stimulant, and cause the absorption, if not entirely, to a great extent, of the opacity. The local treatment must be continued for some little time after the redness has gone, otherwise a recurrence may take place. Calomel dusted into the palpebral aperture is often beneficial in chronic ulcerations ; this practice, however, sometimes causes intense pain, and I prefer the use of the yellow oxide of mercury ointment. The use of applications containing the salts of lead is particularly to be avoided in all corneal ulcerations, inasmuch as a permanent opacity may be formed from the deposit of an insoluble carbonate of the metal.

Counter-irritation is an old and well-known assistant in the cure of chronic forms of ulcer. This may take the form of a blister to the temporal region or behind the ears, the former place being the far better one. The use of setons has almost been given up. A more rigorous method of counter-irritation is that used by Argyll-Robertson.¹ For this purpose, the skin of both lids extending just beyond the orbital margins should be moistened and rubbed with pure silver nitrate, their edges having been made to adhere together by means of boracic ointment. As a result, there is considerable pain for about an hour, followed by œdema of the lids. Boracic ointment should be used as a dressing. The lids are normal in from

¹ *Ophthalmic Review*, 1893, p. 340.

seven to ten days, with no resulting scar. If a simple or phlyctenular ulcer becomes infected, its treatment must be more radical. (See under Suppurative Keratitis.)

iii. The creeping phlyctenular ulcer has received the appellation *fascicular keratitis*. A marginal phlyctenula ulcerates, and the ulcer slowly wends its way towards the centre of the cornea, followed by a leash of blood-vessels. Its growing point is convex in outline, and the ulcer itself is in shape like a new moon, for it heals behind as it progresses, it is yellow in colour and slightly depressed. Hypopyon (*i.e.* pus in the anterior chamber) may coexist. The ulcer may pass right across the cornea, leaving a band of opacity as a permanent testimony of what has taken place. It must not be confounded with riband-keratitis, which has a totally different origin. Ulcers not infrequently appear in eyeballs in which the cornea is densely leucomatous, also in old glaucomatous and other disorganised eyeballs, without any apparent cause. Ulcers secondary to ophthalmia, &c., are dealt with elsewhere.

iv. *Herpes corneæ* is a rare form of superficial keratitis, occurring usually in conjunction with, or following an attack of, herpes ophthalmicus (p. 13), although it may be seen, but more rarely, as a catarrhal form. It is an exceedingly troublesome affection, and recurs persistently. One, two, or more blebs form on the cornea, more frequently upon its lower half, which eventually burst, leaving small ulcers in their place. These corneal changes are attended with considerable circumcorneal injection, slight photophobia and lachrymation. A characteristic feature is the loss of all sense of touch and pain in the cornea. This anæsthesia is very detrimental to the healing process of the ulceration, as the patient is liable to rub the cornea with a handkerchief to 'try and clear the eye,' not aware of the damage that is being self-inflicted. Intense frontal neuralgia is a usual accompaniment of this complaint, and is difficult to relieve. The loss of corneal epithelium is readily shown by the instillation of a drop of fluorescein solution, which stains the denuded substantia propria green, whereas the healthy epithelium remains unstained (F. 18). *Herpes corneæ* has frequently been known to occur during an attack of influenza. Another disease which predisposes to this

condition of the cornea is malaria; such cases are greatly benefited by antimalarial treatment.

Treatment.—Keep the eye bandaged, and allow no one except a nurse or intelligent friend to remove it when the necessary atropine drops are instilled. Cocaine should be avoided, as it is not necessary and only detrimental to the corneal epithelium. Counter-irritation usually intensifies the neuralgia. Tonics, as quinine and strychnine, are beneficial and necessary, as well as a supporting diet. The application of the continuous galvanic current, one pole placed at the nape of the neck and the other over the closed eyelids, appears to relieve pain and hasten the cure.

v. **Dendritic Keratitis.**—The etiology of this comparatively rare form of ulcerative keratitis is still *sub judice*. It is thought by some to be a form of herpes corneæ of the catarrhal form; the numerous small herpetic ulcers run together, having between them narrow bands of more superficial ulceration. A treelike appearance is thus produced. There is, however, no doubt that dendritic keratitis often appears without any previous vesicular stage. Its mode of spreading, by throwing out branches, suggests a fungoid origin. Strong antiseptics, such as pure carbolic acid, or, better still, the actual cautery, are necessary for the cure of this disease.

vi. **Filamentary keratitis** is a variety of vesicular keratitis. It was first described by Leber, and subsequently more fully investigated by Nuel and Hess.

Clinical features.—This disease is recognised by the presence of minute filaments seen lying on the surface of, and attached by a pedicle to, the cornea. There is little or no cloudiness, circumcorneal redness is slight or absent, and the symptoms of most forms of keratitis—viz. photophobia, lachrymation, &c.—are wanting. The filaments, or tags, are seen chiefly on the lower half of the cornea and appear like threads of mucus, but though freely movable they are not swept away by the act of blinking. With a lens they are seen to be attached by a pedicle to the cornea and free, with a small transparent bulbous swelling at the opposite end. They are of various lengths, and in their most elementary form appear as small vesicles. Fluorescein will stain numerous small

areas green where the filaments have become detached from the cornea.

Etiology and pathology.—The actual cause is not known. The nature of the filamentous threads has been variously interpreted. Leber and Fischer thought they were fibrinous, connected with an ulcer, and the result of an herpetic eruption. By some they are considered to be mucus; by others, especially by Nuel¹ and Hess, to be epithelial. This latter view appears most consistent with histological investigation. It is believed that a vesicle is formed on the cornea, that it becomes twisted upon its axis by the action of the eyelids and by repeated torsion to form a delicate cord of twisted epithelium, at the end of which is the original vesicle suspended on the cornea. Under the microscope the filament is seen to be composed of a central twisted cord, not unlike the appearance of an umbilical cord as seen by the naked eye, surrounded by a covering of mucus containing some nuclei. The use of impure salts of atropine has been assigned by Albrand as a cause.

The *treatment* is palliative. The local use of nitrate of silver in solution (2 per cent.), or chloride of ammonium (2 per cent.), has been recommended. It is advisable to stop mydriatics if in use.

vii. **Bullous keratitis** is an inflammation of the cornea associated with the formation of a large bleb, or bulla.

Clinical features.—One large bulla is usually present, though exceptionally two may be seen; it is more often than not situated just below the centre, though it may appear at any part of the cornea. There is circumcorneal redness, cloudiness, or even opacity of the cornea, and severe neuralgic pain, for which the sufferer seeks advice. After a time the bleb bursts, and leaves a large area of Bowman's membrane exposed. The epithelium is soon regenerated, and almost for a certainty a fresh bulla will be formed. The fluid is between the epithelium and Bowman's membrane; it does not appear to be under great tension, as the bulla is often flaccid and pendulous. Apart from other evidence, this variety of keratitis is distinguished from herpetic eruptions of the cornea by the size and single nature of the vesicle.

¹ Nuel, *Archives d'Ophthalmologie*, 1892.

Etiology and pathology.—Bullous keratitis is rarely seen in otherwise healthy eyes. It attacks those disorganised by choroiditis, irido-cyclitis, &c., in which the tension has become raised and staphylomata have developed. It has been known to occur in eyes which have suffered from some slight injury, as a blow from a cork, or some similar accident, and to repeatedly recur with its accompanying ciliary neuralgia. It is believed to be by some a disturbance in the lymph circulation; by others, a dropsical degeneration, or liquefaction, of the deep cells of the anterior epithelium.

Treatment.—The apex of the bulla should be carefully cut away with curved iridectomy scissors, and the exposed surface touched either with a solution of nitrate of silver (F. 1) or with the mitigated silver crayon (F. 7). If the tension is raised, miotics—eserine or pilocarpine—should be instilled, the eye protected with gamgee tissue and firmly bandaged. If the tension keeps up, paracentesis or sclerotomy should be performed. In those cases in which the eye is completely disorganised, excision is the best procedure and affords immediate relief.

Vesicular keratitis is a modified form of bullous keratitis. Instead of a single bulla, a number of minute vesicles occupy, usually, a central horizontal band of the corneal surface. The etiology of this variety is considered to be the same as that of bullous keratitis. Attempts should be made to destroy the vesicles, but they are only too prone to recur, with acute symptoms, and enucleation may be necessary.

viii. **Rodent Ulcer of the Cornea** (Mooren).—The rodent ulcer (in no way allied to rodent ulcer of the skin) is a most serious affection; it commences at the periphery, usually at the upper and lower margins of the cornea, and spreads slowly and intermittently towards its centre. It never perforates, but destroys the superficial layers, and leaves behind a dense opacity. If unchecked the whole cornea is, in time, rendered opaque. This form is usually seen in elderly subjects, and appears as if it had its origin in the arcus senilis. Its cause is unknown. Fortunately, it is a rare disease, and seen more often abroad than in this country. It is frequently bilateral; there is occasionally, though rarely, hypopyon. For treatment, the galvanocautery should be freely used, the apparently normal tissue of

the cornea at the edge of the ulcer being cauterised as well as the floor of the ulcer itself. In spite of this treatment, however, too often the whole cornea is attacked. The prognosis as to sight is, however, not so hopeless as would appear to be the case, since the dense opacity partially clears to a wonderful degree, slight though useful vision being regained.

ix. **Suppurative Keratitis.**—Suppuration in the cornea is the result of septic infection. It presents different clinical varieties, the most common of which is *hypopyon-ulcer*—a suppurating deep or serpiginous ulcer of the cornea, with pus or puro-lymph in the anterior chamber.

Besides hypopyon-ulcer, an abscess of the cornea may be met with. It is a feature of interest how rarely diffuse suppurative keratitis is seen; the pus in the cornea is strictly limited. The anterior chamber is sometimes filled with pus, in which case the whole cornea appears yellow, as if pus had diffused itself between the lamellæ; but a paracentesis of the anterior chamber will usually show how little actual suppuration is taking place in the corneal tissue.

(a) *Deep ulcers of the cornea* are frequently caused by an injury, such as an abrasion, a scratch, or a contused wound of the cornea. An abrasion of the cornea, as a rule, heals with remarkable rapidity; but in those who are broken down in health by senility, alcoholic excess, or disease, the corneal lesion ulcerates and not infrequently suppurates. Deep ulcers occasionally follow the rupture of a pustule, as in phlyctenular keratitis. They are not infrequent complications during an attack of small-pox or after measles. They constitute, as we have seen, a serious feature in severe inflammation of the conjunctiva, be it granular, purulent, or diphtheritic. A predisposing cause is frequently obstruction of the nasal duct, the stagnant lachrymal secretion in the lachrymal sac being the source of septic infection. In all cases, however, there must be a primary loss of corneal tissue.

Deep ulcers commence by first attacking the epithelium, and then spreading both in extent and depth to the substantia propria of the cornea, destroying both the corneal corpuscles and the intercellular tissue. Their edges are copiously infiltrated with leucocytes, and present a greyish-white colour,

which gradually shades off into clear corneal substance. The base is usually irregular, and is of a yellowish colour. When the process has ceased to be progressive, the edges of the ulcer become less abrupt, and its floor is gradually filled by regular layers of cells, which become organised into white fibrous tissue; the epithelium then begins to be restored, and the surrounding corneal tissue regains its transparency. Bowman's membrane is never restored.

Symptoms and complications.—The degree of the usual symptoms of suppurative keratitis is very variable, and often not so severe as in the simple form. There is, on the other hand, frequently œdema and redness of the lids, sometimes making the examination of the eye extremely difficult and painful, and conjunctival chemosis. *Hypopyon* is frequently present. Hypopyon is a collection of pus in the anterior chamber. It is sterile, unless secondarily infected by a perforated ulcer, and is a result of a concomitant iritis, exudation taking place from the iridic vessels. The pus varies in amount and consistence. At first small, it may finally nearly fill the anterior chamber. It may be sufficiently liquid to always occupy the lowest part of the anterior chamber whatever the position of the head, or, on the other hand, it may be so viscid and fibrinous as to be immovable. It is always a grave symptom, implying spread of the disease.

In deep ulcers there is always danger of *perforation*. The posterior elastic lamina is ruptured by the intra-ocular pressure, and aqueous escapes. As a result of the sudden diminution of the intra-ocular pressure, extensive detachment of the retina is liable to occur. The extent of the damage done by perforation depends largely on the position of the ulcer and on the condition of the iris at the time. If the ulcer is central, or if it is nearly central with a dilated pupil, as a rule no harm is done by the perforation. Indeed, the spontaneous rupture of a long-standing ulcer is frequently followed by an immediate acceleration of the healing process.

(b) *Serpyginous ulcers of the cornea* generally occur in elderly or prematurely old people. They are most commonly found amongst those whose occupation exposes them to slight wounds of the cornea. The agricultural classes appear to be

especially liable to this disease, their corneæ being frequently scratched by husks, straw, &c. An abrasion of the cornea is followed by septic infection, the nidus of the germs usually being the lachrymal sac. The specific organism of the disease is the pneumococcus lanceolatus of Fraenkel.

This kind of ulcer may commence in any part of the cornea; though, on account of its usual traumatic origin, the pupillary portion of the cornea is generally first involved. It spreads superficially rather than deeply, and in one direction, towards which it presents a white or yellowish-white crescentic border, which may be to a certain extent undermined. The surrounding cornea is, as a rule, more or less infiltrated. The part first attacked may become filled up by new tissue, whilst the ulceration creeps along the cornea.

A serpiginous ulcer may be comparatively chronic, but it is more frequently acute, being attended with considerable pain, lachrymation, and photophobia. A severe iritis often accompanies it, the exudate from the iridic and ciliary vessels collecting in the anterior chamber—hypopyon.

Treatment of suppurative keratitis.—The treatment of suppurative keratitis differs from that of the simpler non-infected ulcers in degree rather than in kind. A dacryocystitis being so often the cause of infection, attention must in the first place be turned to the condition of the lachrymal apparatus: the entrance into the sac must be made free, its contents purified, and any nasal duct obstruction removed; removal of the sac may be necessary.

Attention must then be turned to the cornea. The palpebral aperture must be repeatedly well irrigated with an antiseptic lotion; atropine, combined perhaps with iodoform, instilled three or four times a day; and belladonna fomentations applied and frequently changed. A useful substitute for belladonna fomentation is the use of continuous dry heat by means of the Japanese muff-warmer. Three leeches should be applied to the temple. At the same time, careful attention to the general condition of the patient must be part of the routine treatment, a purge at the outset being always necessary. Twenty-four hours of this treatment is usually followed by a great change for the better. The hypopyon has

diminished ; the ciliary injection is less marked ; the edges of the ulcer are cleaner, and the cornea in the immediate neighbourhood less infiltrated ; and the area that stains with fluorescin less extensive.

Should, however, there be no improvement, more energetic measures must be undertaken, otherwise a deep ulcer may perforate, a serpiginous ulcer spread over the entire cornea, the hypopyon fill the whole anterior chamber, and a panophthalmitis even be set up. The indications in these cases are a more powerful antiseptic, and evacuation of the aqueous with any inflammatory products it may contain. The antiseptics that we may use are absolute alcohol, pure carbolic acid, and the actual cautery.

Absolute alcohol, the least powerful of the three, is indicated in the more superficial forms of suppurative keratitis, and in the comparatively early stages ; while *pure carbolic acid*, being more virulent in its action, is to be used for the deeper varieties. To make the application, after cocainising the eye, the cornea is to be carefully dried by blotting-paper, and the solution applied by means of a fine splinter of wood or glass. The extent of the ulcer should previously be demonstrated by fluorescin.

The *actual cautery* is, however, the most reliable antiseptic, and its action can be better localised than that of either

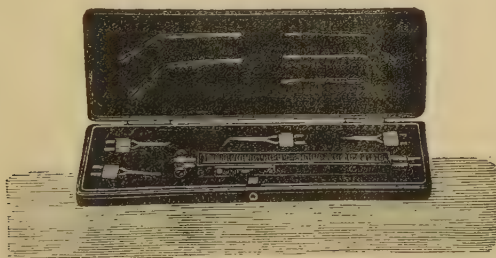


FIG. 32.—Galvano-cautery (one-third size).

alcohol or carbolic acid. A small pointed Paquelin's thermo-cautery may be used ; but a still better instrument is the small galvano-cautery represented in figs. 32 and 33.¹ It is light

¹ Made by K. Schall, 52 Great Marylebone Street, London, W.

and easily manipulated, and is supplied with a variety of small platinum terminals, which are flat, blunt, or sharp, and so can be applied to the surface and beneath the edges of the ulcer in the most efficient manner; and should it be desired to perforate the anterior chamber at the same time, this can



FIG. 33.—Cautery Points (natural size).

easily be effected by the pointed terminal. Five per cent. solution of cocaine instilled for some minutes before the operation suffices to prevent all severe pain.

The object of the actual cautery is essentially to destroy infective micro-organisms which are presumed to be rampant just at the junction of the healthy and the suppurating tissue. The surface should be gently dried with lint before using the cautery, and the latter used at a dull red heat.

Diminution of intra-ocular tension is often indicated, especially in deep ulcers, where there is danger of perforation or protrusion of Descemet's membrane. The intra-ocular tension may be reduced in several ways.

(i) The use of *eserine drops* (F. 17), with the addition of cocaine, every few hours has the combined effect of relieving the tension and stimulating the ulcerated surface. This remedy is strongly advocated by some surgeons; but it must be remembered that eserine is an irritating substance, and so is likely to increase any iritis that may exist; also, that it is a powerful miotic, and thereby favours the formation of extensive posterior synechiæ. When an ulcer is small and *peripheral* and in danger of perforation, or has recently perforated, the use of eserine may prove very useful in drawing the iris away from the affected part; but when the ulcer is *central*, the use of atropine is a much better agent, since it not only soothes the eye, but tends to keep the pupil dilated and the iris away from the perforation if it should occur.

When the iris has actually prolapsed through the perforated

cornea, the protruding portion should be removed as far as possible so as to prevent the iris being clamped in the scar of the cornea. In cases of recent date, say under eight days, the iris can easily be seized with iris forceps, drawn well forward, and cut off close to the cornea with iris seissors, after which it will generally recede into the anterior chamber, and either keep

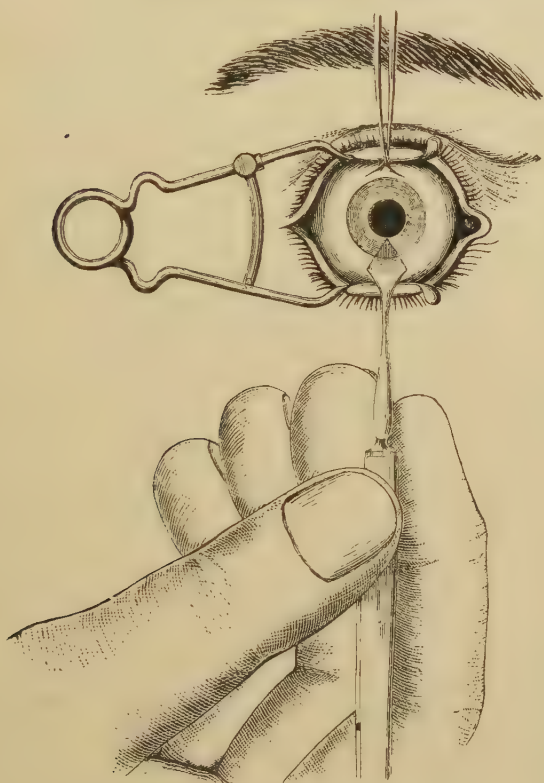


FIG. 34.—Paracentesis of the Anterior Chamber.

free of the wound or only be adherent to its posterior surface. In older perforations this cannot be done; but even here it may sometimes be liberated from its adhesions in the perforating wound and cut off level with the surface of the cornea. In large perforations with prolapse, where the lens also presents itself, its capsule may be lacerated and the lens allowed to escape.

(ii) *Paracentesis of the anterior chamber* is a very excellent means of temporarily reducing the tension of the eye, and is most useful in these cases of threatening or actual perforation. It is superior to the use of eserine, inasmuch as it completely relieves tension, and often gives marked relief to the intense pain which is frequently experienced.



FIG. 35.
Blunt Spatula.

The operation is performed as follows: The eye having been cocainised, the patient is placed in the horizontal posture, and the eyelids separated by a speculum; the eye is fixed by means of fixation forceps in the manner shown in fig. 34. A triangular keratome is then introduced at the lower part of the sclero-corneal junction. Moderately firm pressure is first made in the direction of the *centre* of the globe—that is, at right angles to its surface. As soon as the point of the instrument is seen just within the periphery of the anterior chamber, its direction is immediately changed, so that the blade passes in a plane parallel to and just in front of the iris; it is continued in this direction until the external wound is about 3 mm. or 4 mm. in length. The keratome is then gradually withdrawn, its blade being kept nearer to the back of the cornea than before.

In performing this operation, attention should be paid to the following points: (1) If the incision is made obliquely, and not at right angles to the surface, there is danger of passing the blade of the instrument between the lamellæ of the cornea instead of directly into the anterior chamber. (2) The direction of the instrument must be changed as soon as the point has entered the anterior chamber, otherwise there is danger of wounding the iris and the crystalline lens. (3) During the withdrawal of the keratome, its blade should be still more approximated to the cornea, as, with the escape of the aqueous, the lens and iris frequently bulge forwards. (4) Should the iris protrude through the wound, it must, if possible, be returned by means of a blunt spatula (fig. 35), or, if this cannot be accomplished, the hernia must be seized with the iris forceps and cut off as in the operation for iridectomy.

(iii) *Saemisch's section* was formerly much used for relieving tension, and at the same time giving free exit to pus in cases of hypopyon. It consists in completely dividing all the affected structures, as well as Descemet's membrane. This remedy, however, is gradually dropping out of use, its place being taken by the actual cautery.

Subconjunctival injections have been used for some time for corneal ulceration, and especially for the suppurative forms. For this purpose cyanide of mercury of the strength 1 in 5,000 is injected subconjunctivally. It was found by Darier that if a few drops of acoine (1 per cent.) are injected with the mercury salt, and if the injection is made as far from the cornea as possible, pain is practically absent. The injection should, if necessary, be repeated every third or fourth day, 10 min. being used on each occasion. Excellent results have been obtained, especially if the ulcer is touched with the galvano-cautery at the same time.

(c) A *corneal abscess*, though not strictly a form of superficial suppurative keratitis, is so closely related in its symptoms and treatment to the deep and serpiginous corneal ulcers, that it will be considered in this place.

A corneal abscess is a circumscribed collection of pus in the substantia propria of the cornea. It is the result of injury or a phlyctenule with sepsis. The injury may be so slight as to have escaped notice. The abscess is seen as a yellow spot or patch in the cornea, surrounded by a grey zone of infiltration. The epithelium is often steamy or lustreless over it. The ocular conjunctiva is more or less injected, often intensely red, resembling raw beef, and chemosis is generally present. In severe cases, pus will be found in the anterior chamber (hypopyon), and the iris acutely inflamed. The abscess usually bursts externally if left to itself. If small, the resulting ulcer will heal without much trouble; but if deep or extensive, Descemet's membrane will protrude as a small transparent vesicle (keratocele) in the centre of the ulcer. Such a condition will not heal without perforation. The symptoms are not, in many instances, very marked; even photophobia is often absent. There is deep-seated or orbital pain, owing, no doubt, to the spread of inflammation to the capsule of Tenon.

Onyx is a term applied to a collection of pus or puriform matter in or behind the cornea, which has an upper convex outline. The condition has received this name from its fancied resemblance to the lunula of the finger-nail. The appearance is attributed by some to the lamellar arrangement of the substantia propria. It is thought by them that pus is diffused between the lamellæ and gravitates to the lower part of the cornea. This view has been objected to by Fuchs on the ground that he had not found this condition in the anatomical examination of such eyes. He discovered that a thin layer of puro-lymph had applied itself to the posterior surface of the cornea in such a manner as to present an upper convex margin, and at the same time to leave a space between it and the anterior surface of the iris. Moreover, from its plastic nature it does not alter its position when the head is inclined towards the shoulder. Pus gravitates to the bottom of the anterior chamber, has a horizontal linear limitation at its upper part (see fig. 11, opposite p. 118), and, being fluid, alters its position with the movement of the head. In rare instances, I feel convinced that pus does diffuse itself between the lamellæ. The quantity of pus in cases of hypopyon is variable: sometimes only a faint yellow streak can be seen at the extreme lower limit; in other cases the anterior chamber may be full, giving the whole cornea a uniform yellow appearance. Hypopyon is rare, except in cases of septic injuries and corneal ulcers; it is, however, seen with gummatous iritis, or even with severe plastic iritis without new-formations.

Etiology and pathology.—The essential cause of this, as of all forms of suppurative keratitis, is septic infection. Pyogenic organisms enter through an abrasion or punctured wound, however small it may be. Staphylococci are sometimes to be found in the pus removed from the anterior chamber, although Leber has shown that suppuration can take place in the anterior chamber in the absence of organisms. In a corneal abscess the pus is not usually fluid, but consists of a somewhat tenacious puro-lymph mixed with broken-down corneal tissue. Infection may be caused by a scratch from a finger-nail or any foreign body; moreover, any slight abrasion

or wound by operation is very prone to be infected if there is suppurative dacryo-cystitis present.

Almost without exception the sufferer is in a debilitated condition. Women run down in health from prolonged lactation, persons senile and decrepid with ill-health or prematurely aged from excessive indulgence in alcohol and other abuses, are subjects likely to suffer. In children in a weak state, phlyctenules may suppurate and cause deep abscesses in the cornea.

Treatment.—A small septic focus will often yield to fomentations, boric acid, poppy-head, or belladonna (F. 24). If belladonna fomentations are not used—which, in my opinion, are the best—it is necessary to supplement the others with the local use of atropine, for the pupil must be kept dilated. If after twenty-four hours there is no visible improvement, the small abscess must be destroyed with the point of a galvano-cautery, under cocaine or a general anæsthetic. If the abscess is a millimetre or more in diameter, it is advisable to destroy its anterior wall with the cautery without delay, and allow of the escape of the débris; and if hypopyon coexist, a paracentesis of the anterior chamber should be performed at the same time. If the abscess has burst and Descemet's membrane protrudes, it should be cauterised and the aqueous humour liberated, for a keratocele prevents healing and invariably ruptures before healing ensues. An extensive suppuration in the cornea should be treated by the method of Saemisch (*vide* p. 135).

Constitutional treatment is as necessary as local treatment. Rest in bed, a nourishing diet, stimulants—such as port wine, champagne, &c., if necessary—and a tonic consisting of ammonia and bark, quinine and iron, or strychnine. The bowels should be freely opened as a preliminary step in the treatment.

x. **Neuroparalytic Keratitis.**—Occasionally we find ulceration of the cornea supervening in cases of paralysis of the fifth nerve, which supplies the trophic and sensory fibres to the eyeball. It is a most destructive kind of inflammation; the cornea may necrose and slough away, with loss of the eyeball. There is, as a rule, anæsthesia of the cornea and conjunctiva. The inflammatory symptoms of ulceration, such

as photophobia, lachrymation, pain, and redness, are not pronounced. In those cases that improve, the process of recovery is most tardy.

The existence of neuroparalytic keratitis has been doubted by some physiologists, who state that the trigeminal nerve has no trophic fibres, but that the corneal ulceration is due to foreign bodies or to injuries, of whose existence the anæsthetic cornea is ignorant. These authorities say that if the eye was covered no lesion would take place, and bring forward experiments on rabbits to prove this. But many cases of insensitive corneæ which have not sloughed are on record; and the insensitive cornea of glaucoma very rarely sloughs, and often remains quite transparent. Besides, neuroparalytic keratitis may exist without loss of sensation.

In the case of ulcer from nerve-lesion, the eye should be closed by means of a light compress and bandage. A little atropine should be used daily to prevent iridic adhesions, while the affection of the nerve is treated by the primary galvanic current, iodide of potassium, and other remedies.

xi. Keratitis the result of Exposure, or Keratitis e Lagophthalmo.—Anything which prevents the normal blinking movements of the eyelid is liable to cause this condition. Desiccation of the superficial layer of the cornea takes place, leading, as the process continues, to a superficial ulceration. If no treatment is resorted to, and sometimes in spite of all treatment, perforation of the cornea takes place, followed by iritis, and finally panophthalmitis, necessitating enucleation. The cause and treatment of this form of keratitis are those of lagophthalmos (p. 23).

xii. Secondary Superficial Keratitis.—Corneal ulcers may be secondary to catarrhal, granular, or purulent conjunctivitis; to entropion, trichiasis, or distichiasis. In these cases, attention to the conjunctival or palpebral affection becomes of the greatest importance; but it must be remembered that irritating substances, such as nitrate of silver and sulphate of copper, if used, must as far as possible be kept away from the ulcer of the cornea—indeed, blue-stone had better not be used at all in such cases; the same remark applies to the use of lead-lotions, for fear of metallic deposit (see p. 164).

Prognosis in corneal ulcer is frequently very difficult both as to the duration of the case and the visual defect which may result. Small dense opacities are generally less deleterious to vision than slighter nebulous conditions which extend over a larger surface. One may, therefore, be less anxious about a small somewhat deep ulcer than about one which is larger and superficial. The nearer to the corneal centre an ulcer is situated or seems to be progressing, the greater the danger of future visual trouble.

When vascularity can be seen at the edges or upon the surface of an ulcer, it may be taken as a sign of repair, and that the destruction of corneal tissue is at an end.

When an ulcer has extended as far as the limbus conjunctivæ, it seldom progresses into the sclera.

After extensive suppuration of the cornea, such as sometimes accompanies purulent ophthalmia and abscess, there often remains some peripheral portion of the cornea which is still transparent and can be rendered useful by a well-chosen iridectomy.

SEQUELÆ OF CORNEAL ULCER.

(a) **Corneal Opacity.** — In the majority of cases, the position of the healed ulcer is marked by a persistent patch of opacity. The density of this presents every shade of variety: when very slight, so as to be perceptible only on close examination, it is called a *nebula*; when distinctly opalescent, a *leucoma*. The opacity is due to the substantia propria of the cornea being replaced by fibrous tissue.

If a patch of opacity thus established happens to be opposite the aperture of the pupil, there is necessarily much interference with vision; if situated nearer the periphery, this is less marked; but in either case it often happens that after cicatrisation the cornea is not restored to its normal thickness, but has a faceted appearance, or even a depression corresponding to the position of the ulcer. The irregularity of surface is in itself sufficient to cause serious diminution of vision, on account of the irregular astigmatism that is produced.

The treatment of corneal opacities depends on the length

of time they have existed, and on their density. As soon as active ulceration has ceased, and all redness of the eye disappeared, atropine must be replaced by an ointment of yellow oxide of mercury (F. 39). This should be used night and morning as a massage, over a period of several months, until the opacity shows no further sign of absorption. The younger the patient, the greater the benefit derived by this treatment. Old nebulae of the cornea have been subjected to many and varied methods of treatment with little or no success. Of these the following may be mentioned: the use of steam by means of an atomiser, electrolysis, subconjunctival injections of antiseptics or aseptics, and grafts from corneae of animals. So far, it must be confessed that this last method of treatment has not been followed by much success. The cornea of one rabbit has been transplanted on to the eye of another, both in France and Germany, by Münck, Königshoffer, Desmarres, and others, but always with the result, that although union might take place between the new and the old tissues, yet the new cornea became shrunken and opaque. The cornea of the rabbit and other animals has also been transplanted to the human eye by Pluvier, Power, and others, with similar results.

When the opacity is dense, and situated in front of the pupillary aperture, but not involving the whole extent of the cornea, the vision may be very much improved by the formation of an *artificial pupil*. For this purpose that part of the cornea which is clearest and most regular in curvature should be chosen. In order to ascertain the position best suited for this operation, the pupil should be dilated with atropine, and the eye examined by the oblique focal illumination (p. 109), and by the ophthalmoscope. With the former, any nebulous opacities will appear as a greyish haze, and any facets or depressions will be directly seen; with the latter, on tilting the mirror in various directions at 20 to 40 cm. without a lens, the red fundus-reflex is interfered with by the appearance of dark patches of the cornea. The methods of operating for artificial pupil are described under the head of Iridectomy.

When the opacity of the cornea is only slight (nebula), it can still be penetrated by rays of light, but, as these are distorted, and thus interfere with the images formed by rays

passing through the clear portion of the cornea, the optical effect of an artificial pupil is unsatisfactory. In such cases, to make an artificial pupil alone is useless, but great improvement is often obtained by rendering the nebula completely opaque by tattooing; then, if the nebula is of large size, an artificial pupil may still further assist vision.

The probable effect of an artificial pupil may be ascertained by dilating the pupil with atropine; if the distant vision is improved by this an artificial pupil will be still more beneficial; if, on the contrary, the distant vision is confused, the operation would probably cause confusion also.

When the opacity is not central, the vision may be but little interfered with, but here the appearance of the eye might also be improved by tattooing the leucoma.

The operation for tattooing the cornea.—The eye must be anæsthetised by the introduction of a few drops of cocaine solution (5 per cent.) for a few minutes before operating. The eyelids are separated by a speculum, and the globe held in position by a pair of epilation forceps. An assistant should hold a small sponge firmly against the upper and outer side of the globe to prevent any tears running over the cornea during the operation. Indian ink, carefully rendered aseptic, is made into a thick paste, and a drop placed over the opacity. The portion to be tattooed should then be well covered with punctures or scratches, by using a grooved needle such as is shown in fig. 36. Perhaps even better than a grooved needle is an ordinary cataract-needle, the punctures being made with the instrument almost parallel with the corneal surface.

Except in the case of large leucomata, a single sitting will usually suffice.

It is sometimes advisable to tattoo also the periphery of the cornea opposite the artificial pupil, so as to prevent the entry of rays through the part, which would otherwise cause some blurring of the retinal image from spherical aberration.



FIG. 36.
Tattooing
Needle.

(b) *The formation of a Keratocele.*—The progress of a deep ulcer of the cornea may be hindered by the resistant membrane of Descemet. This is caused to protrude through the ulcer by the intra-ocular pressure, and may form a small clear

vesicle above the surface of the cornea. It is called a keratocele (see fig. 37). With its formation the process of ulceration may stop, cicatrisation occurring, and the clear keratocele persisting in the centre. More frequently, however, a keratocele is the prelude to complete perforation of the cornea. In these cases, it should be touched with the galvano-cautery, atropine instilled, and a firm pressure-bandage applied.

(c) **Perforation** of the cornea may or may not be preceded by a keratocele. It is the worst complication of a corneal ulcer that may arise, and consequently its prophylactic treatment is of extreme importance (see p. 134). As has been said, perforation of the cornea may be the best thing that could happen in a case of a deep corneal ulcer. Improvement immediately begins, and continues with no untoward results. In these simple cases, atropine and a pressure-bandage, with rest in bed, are all that are needed to aid Nature in the work of repair. Much more frequently, however, further complications may arise—namely, anterior synechia and anterior staphyloma, corneal fistula, retinal detachment, anterior polar cataract, iritis, and panophthalmitis.

(d) **Anterior synechia**, or adhesion of the iris to the cornea, is caused by perforation of the latter, either from disease or injury. The anterior chamber being thus emptied of its aqueous humour, the iris is pushed forward so as to come into contact with the perforation, inflammatory exudation takes place, and the iris becomes adherent either to the posterior surface or in the depths of the cicatrix, the resulting condition being termed *leucoma adhærens*.

The *symptoms* and the *consequences* of anterior synechia vary in proportion to the extent of the lesion. In slight cases, where there is only an adhesion of a portion of the pupillary edge of the iris to the posterior surface of the cornea, there may be but little inconvenience; the vision, however, is usually more or less defective, and, the movements of the iris being limited by the synechia, the patient is liable to attacks of iritis, pain, &c.

When the iris is entangled in the cicatrix, it shows itself as a black patch in the cornea. The vision here is generally deficient, and although sometimes there is no great incon-

venience except that of the loss of vision, yet these cases are liable to attacks of severe pain in and around the eye, to recurrent iritis, and even to panophthalmitis.

When the iris protrudes through a perforation, and becomes adherent in that position, there is frequently at first a leakage of the aqueous humour from the exposed surface; as contraction of the cicatrix goes on, however, this leakage lessens, and the surface of the iris becomes finally covered with a layer of lymph. The organisation of this lymph may so stop the filtration as to increase the intra-ocular tension. (See Secondary Glaucoma.)

(e) **Anterior staphyloma** (fig. 37) signifies a bulging forwards of the whole or part of the anterior surface of the eyeball beyond its normal curvature, excluding keratoconus or conical cornea (see p. 157).

Anterior staphyloma is almost invariably the result of perforation of the cornea, either from ulceration or from injury. There are two forms of anterior staphyloma. In the commoner variety, the apex of the staphyloma is composed of transformed iridic tissue; the

remainder of the bulging is partly corneal, partly iridic, the latter predominating. There is usually complete absence of the anterior chamber. The mode of formation of this variety is as follows: As soon as perforation takes place, there is immediate escape of the aqueous humour, and, as we have just seen, the iris comes forwards in contact with the opening, and may protrude through it; inflammation then takes place from exposure, and the parts become matted together by exudation, so as to fill up the orifice. The cicatrix, however, being weaker, is unable to resist intra-ocular tension,

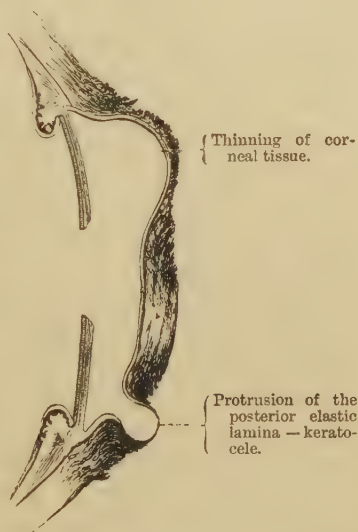


FIG. 37.—Staphyloma of Cornea.
(After Stellwag von Carion.)

which is now re-established by the closure of the perforation, and bulges forwards. The extent of this deformity presents every degree of variation: there may be merely a small bladder-like protrusion of iris; or the greater part of the cornea may have sloughed off, with protrusion of a large surface of altered iris; the extent of the projection is sometimes so great as to prevent complete closure of the lids. This condition of bulging of the cornea and adherent iris is liable to set up serious trouble, not only of these structures, but also of the neighbouring structures in the ciliary region, which may lead to complete disorganisation of the globe.

In the second and rarer form (see fig. 37), there is no prolapse of iris, but merely a bulging forwards of corneal tissue at the seat of the cicatricial tissue. The structure of the cornea becomes much altered, the epithelial layer is thickened, the substantia propria is thin, opaque, and of a grey or yellowish colour. In this form the anterior chamber is deepened. Vision is impaired in proportion to the extent and position of the corneal surface affected.

Treatment.—Directions have already been given under the head of Ulcers of the Cornea for the prevention of staphyloma. When once fully formed, it is far from amenable to treatment.

In small, partial, and recent cases the compress should be continued, and the intra-ocular tension diminished by *paracentesis* of the anterior chamber; by repeating this every second or third day for a few times, the cicatrix often gains strength and becomes stationary. Should the tension not be sufficiently diminished by this means, or should it become increased above the normal, more benefit would be derived by excising a portion of the iris (see Iridectomy). A good large iridectomy should be performed opposite the clearest portion of the cornea. This would permanently relieve the tension, and an artificial pupil would be at the same time established.

When the staphyloma is small and circumscribed, some portion of the cornea remaining sufficiently clear for useful vision, the projection may with advantage be excised and an artificial pupil at the same time made by a small iridectomy behind the clear cornea. The excision may be performed

either by seizing the projection with forceps and cutting it off with curved scissors, or by using the corneal trephine as for conical cornea. This mode of procedure often results in a sufficiently firm cicatrix.

When the whole cornea is involved, the eye often becomes the seat of severe pain, and the increased dragging of the iris upon the ciliary region causes its disorganisation; the lens becomes opaque and perhaps dislocated. The staphyloma may be so large as to be unsightly, and to prevent proper closure of the eyelids. When such is the case, it is necessary to remove the protruding portion, not only on account of its unsightliness, but because, by constant exposure and irritation from the partly closed eyelids, it would undergo ulceration and sloughing, with escape of the contents of the globe. It may be removed (1) by abscission, (2) by abscission with evisceration, (3) by abscission with evisceration and the introduction of a glass or metal globe, or (4) by enucleation. (See p. 146.)

(f) **Corneal Fistula.**—In rare cases the perforation remains open, with the iris adherent to its margins, the anterior chamber being necessarily absent. A flap of conjunctiva should be transplanted over the fistula, being kept in position by a few fine sutures.

(g) **Retinal Detachment.**—The sudden diminution of intra-ocular tension which occurs at the moment of perforation may cause sudden effusion of blood or serous fluid into the post-retinal space, producing thereby complete detachment of the retina. These cases usually end in enucleation or one of its substitutes.

(h) **Anterior Polar Cataract** has as its commonest cause perforation of the cornea as a result of ulceration; but see p. 368.

(i) **Iritis and panophthalmitis** may succeed the perforation of a corneal ulcer, owing to the direct spread of the infective material. As a rule, enucleation is necessary.

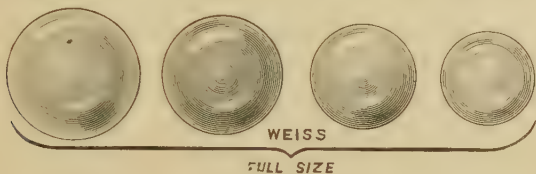
THE OPERATION OF ENUCLEATION AND ITS SUBSTITUTES.

Abscission consists in removing the anterior part of the globe by incision of the sclera at a distance of 2 or 3 mm. from the corneal margin. It dates from very early times, having been practised by Celsus. The globe is fixed by forceps in the usual way, and a Beer's knife is used to incise the upper half of the sclera and ciliary body; the lower half is then excised with scissors. The iris and part of the ciliary body, the lens, and such of the vitreous body as may be presenting, are also removed. In former days the eye was left in this condition until the extensive opening became filled with granulations and finally healed up. In order to accelerate the healing of the wound and to improve the shape of the stump, the use of sutures was introduced by de Wecker and G. Critchett. Owing to the liability of sympathetic irritation and inflammation supervening after this operation, it has recently been abandoned and succeeded by the following.

Evisceration (exenteration) was probably first performed by Noyes in 1874, but it was not until 1884, when Alfred Graefe began to practise it with the view of preventing death from meningitis in cases where suppuration and panophthalmitis were present at the time of the enucleation of an eye, that ophthalmic surgeons generally began to use it. It consists in first removing the cornea by means of an incision at the sclero-corneal junction, the upper incision being made with a linear cataract-knife and the lower completed with scissors; then the whole contents of the remaining cavity of the sclera are removed by means of a steel scoop. Great care must be exercised to thoroughly remove all traces of the choroid, which can be peeled off from before backwards as far as the optic disc before much scraping is required. Dry lint will be found useful in stopping hæmorrhage and clearing away remaining fragments of choroid, which are seen as brown or black patches. It is often very difficult to illuminate the interior of the globe when eviscerating it in this manner, both owing to its shape and the necessary oozing of blood from the retinal and short ciliary arteries. I find a small electric lamp very useful in

overcoming this difficulty. Strict antiseptic rules should be followed, and the parts well irrigated with a solution of corrosive sublimate 1 in 5,000, before and after closing the wound. Finally, the margins of the wound are drawn together by a few sutures.

Mules's Operation.—A modification of the above operation was about the same time introduced by Mules, with the object of providing a still better stump upon which an artificial eye could move more freely and appear less sunken in the orbit.



FIGS. 38, 39, and 40.—Mules's Artificial Vitreous, Introducer, and Evisceration Scoop.

The operation consists in removing the cornea and the contents of the sclera, and introducing an 'artificial vitreous' into the scleral cavity in the form of a hollow glass ball (fig. 38). It is performed as follows: The cornea is removed at the sclero-corneal junction as in simple evisceration. All the contents of the globe are separated from the sclera from before backwards by a special steel evisceration scoop (fig. 40), or a small Volkmann's spoon, and finally removed. No shreds of choroid or ciliary process must be left. The hæmorrhage is easily controlled by hot lotion. Then, by means of a special

introducer (fig. 39), the artificial vitreous is inserted into the cavity. The size of the ball must be considerably smaller than the cavity it is destined to occupy ; it may, however, be necessary to enlarge the opening by two horizontal or vertical snips. Vertical or horizontal sutures, usually five in number, are introduced, silk being on the whole preferable to catgut. A light but firm antiseptic dressing is applied, and over it an ice-bag. Mules provides for drainage of the orbit by introducing a gold wire to the back of the globe from the outer canthus ; it is left *in situ* for at least three days ; this, however, appears to be quite unnecessary. There is generally, in my experience, a considerable amount of pain, chemosis, and swelling of the eyelids immediately after the operation ; the ice-bag is very valuable in diminishing these symptoms ; they usually subside in the course of six or eight days. In some cases suppuration sets in, especially where the selected glass globe is too large ; in such a case there may be no union of the scleral wound at all, or it may open up afterwards and give exit to the glass ball, so that the condition of the eye is similar to that following simple evisceration.

The success of the operation depends on the careful selection of cases, the choice of a sufficiently small glass globe, the completeness of the evisceration, the almost complete stoppage of the hæmorrhage before the globe is introduced, and on rigid antisepsis. Sometimes, after an interval, perhaps, of some years, the cicatrix in part breaks down, the glass ball becoming visible. Should this occur, the edges of the opening should be pared, and a graft taken from the mucous membrane of the lip applied.

When all goes well with the operation, the cosmetic result is all that could be desired, and the artificial eye moves freely in almost every position of regard. The danger of broken fragments of glass resulting from any severe blow upon the eye appeared to be great, but no cases of the kind have hitherto been recorded. On account of this supposed danger, celluloid and silver balls have been used. With the latter, several instances of argyriasis have been recorded.

The original operation differs somewhat from the above description. Mules divided conjunctiva and cornea separately,

and dissected up the former as far as the equator of the eyeball. Similarly, he used a double set of sutures—first, vertical ones for the sclera; and, secondly, horizontal ones for the conjunctiva. The simpler method is in general use at the present time.

Enucleation of the Eye.—Removal of the eyeball appears to have been first proposed in 1841, and first performed by Stoeber of Strasburg in 1842.

Operation.—The patient must be fully anæsthetised. The operator stands behind the patient's head. The instruments required are speculum, fixation forceps, straight and curved scissors, and strabismus hook (figs. 41 to 44). The eyelids are widely separated by the speculum, and the globe is held steady by seizing the conjunctiva with the forceps near the margin of the cornea.

The conjunctiva is then divided all round, and close to, the cornea, leaving only sufficient for the forceps to hold on by; the capsule of Tenon is at the same time opened by carrying the deeper blade of the scissors well beneath the conjunctiva close to the sclerotic.

The strabismus hook is then passed into Tenon's capsule and successively glided beneath the tendon of each muscle, which is divided with the scissors between the hook and the globe. The speculum is allowed to open more widely by loosening the screw, and is pressed slightly backwards; the globe then starts forwards, and protrudes through the palpebral aperture. The scissors are now introduced either at the inner or outer canthus, having their concavity towards the globe. As they reach the back of the eye the optic nerve is felt for. The scissors are then slightly withdrawn until they can be opened, with the nerve between the blades. They are then pushed in and the nerve divided. One or two more snips are now required to sever any remaining tissues, and the globe is removed. One now observes a smooth cavity, corresponding to the position of the eye, which is lined by the capsule of Tenon and is bounded in front by the detached ocular conjunctiva. The speculum is then taken out. Hæmorrhage is easily stopped by syringing the cavity with hot perchloride lotion. Plugging with antiseptic gauze may be necessary.

Immediately over the closed eyelid is placed a piece of Turkey sponge, and over this a tight compress of several layers of cyanide gamgee tissue. The eye should be dressed in twenty-



FULL SIZE.

FIG. 41.
Squint Hook.FIG. 42.
Speculum.

FULL SIZE

FIG. 43.
Fixation Forceps.FIG. 44.
Curved Scissors.

four hours, and the sponge removed. Recovery is rapid, the patient being convalescent in three or four days.

In excising an eye, care must be taken not to puncture the globe, as the flaccidity caused by the consequent escape of vitreous renders the completion of the operation more difficult than when the globe is intact. The presence of old or recent inflammatory adhesions often renders excision difficult. In this case, if it is found that the hook cannot be passed under the tendons, the adherent tissues must be carefully dissected away from the globe with the scissors alone. When an eye is excessively large and elongated, as happens in some cases of buphthalmos and myopia, it is very difficult to divide the optic nerve without cutting the sclerotic at the posterior pole of the eye. Should this accident occur, and the back of the globe be left in the orbit, it should be removed at once.

Some surgeons prefer to bring the edges of the conjunctiva together by fine silk sutures after excision.

Frost has, in many cases, successfully inserted into the capsule of Tenon an artificial vitreous of the kind used by Mules (see p. 148), suturing the capsule and conjunctiva over it in separate layers.

Arlt's method of enucleation is quicker than the foregoing, and is preferred by some surgeons. It is performed as follows: First, an incision is made through the conjunctiva over the insertion of the internal rectus; the tendon is exposed and divided near its insertion. The small portion left upon the sclera is then seized with the fixation forceps, and thus used to rotate the globe in any required direction until the completion of the operation. From the conjunctival wound thus made the conjunctiva and capsule of Tenon are divided with the scissors, first below and then above the corneal edge. Next, the inferior rectus is divided by passing one blade of the squint scissors beneath its tendon and the other blade above it, and cutting as close to the sclera as possible. Then the superior rectus is divided in a similar manner. This being done, the closed scissors are passed from the inner canthus behind the globe until the optic nerve can be felt; the globe is at the same time drawn forward by the forceps attached to the internal rectus, so that the optic nerve is somewhat tense. When the optic nerve

is thus felt the scissors are opened and the nerve divided as close to the globe as possible. After the division of the nerve, the globe will be more freely movable in every direction; the obliqui and external rectus must now be divided close to the sclera, and the operation is complete. No squint hook is used.

When the operation of enucleation has been aseptically performed, the union of the wound usually takes place by first intention. The cavity formed by the removal of the eye is enclosed posteriorly by Tenon's capsule, and is bounded in front by the ocular conjunctiva, which has been dissected from the globe. In the posterior part of the stump thus formed is situated the optic nerve; with its sides the tendons of the ocular muscles become united, and its anterior part becomes closed in by the conjunctiva.

When suppuration follows the operation, it must be concluded that there has been infection of the wound either from the palpebral sac or from some other source.

Complications and sequelæ that may follow the operation of enucleation.—(a) *Meningitis*.—A few cases have been recorded in which enucleation, performed whilst the eye was in a condition of suppurative panophthalmitis, has been attended with a fatal result owing to the supervention of suppurative meningitis. It is therefore a rule with some surgeons not to enucleate a suppurating eye, but either to wait until the process has subsided or to perform abscission with evisceration.

In my own practice, I should not hesitate to enucleate a suppurating eye provided that strict antiseptic precautions were observed and the after-dressings were loosely applied, so that free exit of infecting organisms could be obtained.

(b) *Hæmorrhage*.—It may be that, as a result of a desire to clinch the optic nerve as far back as possible, the ophthalmic artery is divided, leading to severe hæmorrhage into the capsule of Tenon. A kind of traumatic aneurysm is thus produced, and must be treated by evacuation and ligature, though the latter often presents considerable difficulty.

(c) *Hæmorrhage from nose or mouth*.—Cases have occurred where, soon after an enucleation, the surgeon has been recalled to treat one of these conditions. It appears that in

these cases the lachrymal bone has been fractured by the scissors during the neurotomy, producing hæmorrhage into the mouth or nose.

(d) A frequent sequela of enucleation is the formation of a button of *granulation tissue* at the apex of the socket. This produces a persistent mucoid discharge, and should be removed with scissors, its base being touched with lunar caustic.

(e) *Bands of fibrous tissue* may slowly form in the socket, rendering it less deep, and often precluding the possibility of wearing an artificial eye. These cases are extremely difficult to treat, since operative interference often results in the formation of still more fibrous tissue.

Optico-ciliary neurotomy was first suggested by von Graefe in 1857. The operation has been by no means extensively practised. It is occasionally performed for the relief of pain in blind, painful, glaucomatous eyes.

Optico-ciliary neurectomy was first performed by Schweigger in 1885 as a substitute for neurotomy, but is only rarely performed at the present time, and by few surgeons.

Indications for Enucleation or one of its Substitutes.—In 1896 a special committee of the Ophthalmological Society of the United Kingdom was appointed ‘to consider the relative value of simple excision of the eyeball, and the operations which have been substituted for it.’ A short *résumé* of its conclusions will be given here.

1. *Intra-ocular malignant growths.*—Enucleation is the only operation suitable. Tumours occupying the posterior part of the eye, such as glioma of the optic nerve or retina, or sarcoma of the choroid, are very apt to travel along the optic nerve towards the brain. In removing eyes thus affected, as much as possible of the optic nerve should, therefore, be removed.

2. *Suppurative panophthalmitis.*—There is an undoubted, though very slight, risk of fatal meningitis where enucleation has been performed for suppurative panophthalmitis. Whether the substitutes for this operation are safer in this respect is not known.

3. *Wounds of the eye likely to excite sympathetic ophthalmitis*.—Mules' operation is probably as safe, from a sympathetic ophthalmitis point of view, as enucleation, provided it be performed within three weeks of the original injury, and, in my opinion, provided there are no signs of irido-cyclitis in the injured eye. Enucleation is to be performed rather than evisceration if sympathetic ophthalmitis has already set in.

4. *Anterior staphyloma*.—Mules' operation is especially indicated in this condition. Abscission is liable to result in a painful stump, and to cause sympathetic irritation.

5. *Shrunken eyeballs*, if painful, should be enucleated, and not eviscerated.

6. *Painful blind glaucomatous eyeballs*.—It is for this class of cases that optico-ciliary neurotomy or neurectomy has been especially performed. The relief of pain is, however, by no means permanent, and Mules' operation is much more satisfactory.

To the above I would add the following indication :

7. *Hæmorrhage* from the fundus oculi, whether owing to an operation or to accidental wound of the globe, which cannot be stopped, must be treated by enucleation or evisceration.

The question of removing a wounded eye will be found discussed under the head of Sympathetic Ophthalmitis (p. 217).

Artificial eyes are made of glass, and are kept in great variety as to size and colour by the best opticians. When the cicatrix of the conjunctiva and other tissues of the orbit is firm, quiet, and free from ulceration or discharge, it is ready to receive the artificial eye ; this condition is usually established in from four to eight weeks. The eye should not be worn continuously. For the first few weeks it may be worn a few hours daily ; after that, if no irritation is experienced, it can be worn all day, but never during the night. The artificial eyes in ordinary use require to be renewed about every nine to twelve months, as they are apt to become rough, and therefore irritating to the conjunctiva.

Celluloid is now sometimes used as a substitute for glass in the manufacture of artificial eyes. The eyes made of this substance are unbreakable, and are lighter than the glass eyes. The edges can be cut with an ordinary penknife to adapt the

eye to any peculiarity of the stump. In appearance they exactly resemble those made of glass, and they are said to be more durable.

The shape of the artificial eye should vary according as enucleation, simple evisceration, or Mules' operation has been performed. In the last case, the old shell (fig. 45, *a*) should be used. After evisceration, however, the new *improved artificial eye of Snellen* is to be preferred. The shell is replaced

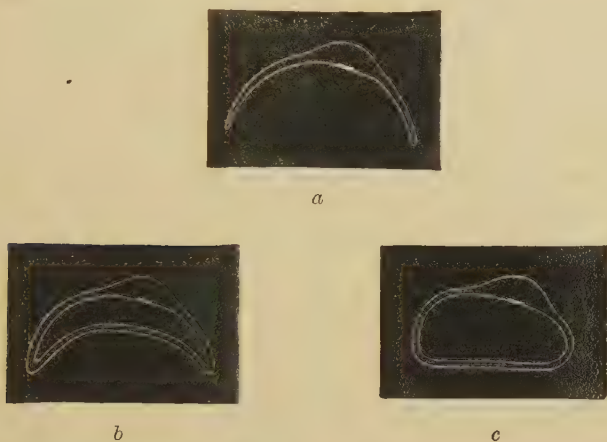


FIG. 45.—Snellen's Improved Artificial Eyes.

by a hollow globe, which gets rid of the large concavity and the sharp edges. Two shapes are used—a double-walled shell (fig. 45, *b*) for simple evisceration, and a larger hollow globe (fig. 45, *c*) for enucleation. These largely do away with the sunken appearance, and greater motility is obtained. The *insertion of an artificial eye* is very easy, and is soon learned by the patient. It must first be steadily pushed beneath the upper lid, and held there whilst the lower lid is brought round its lower edge. *Its removal* is still more simple. The lower lid is depressed so as to expose the lower edge of the eye, and beneath this a probe is placed, by which the eye is brought forward. It then slips out by its own weight, and should be caught in a handkerchief held for its reception.

DEGENERATIVE CONDITIONS OF THE CORNEA.

Arcus senilis is a hyaloid degeneration of the superficial layers of the cornea in the neighbourhood of the sclero-corneal margin, but it is always separated from the limbus by a perfectly clear ring of corneal tissue. Usually occurring after middle age, it is not unknown in children, when the condition is often hereditary. It first appears at the upper and lower margins of the cornea as a more or less milky-looking arc, the whole circle being gradually completed.

Ribbon-like keratitis, or zonular opacity of the cornea, usually occurs in blind and insensitive eyes, especially as the result of glaucoma. A primary form is occasionally found, and is thought by von Graefe to foreshadow the onset of glaucoma. A horizontal band of the cornea, corresponding to the exposed interpalpebral area, is chiefly affected, the appearance being that of a band composed of innumerable minute dots. The condition is due partly to a hyaloid degeneration of Bowman's membrane and the superficial layers of the substantia propria, and partly to the deposition of lime salts in the same structures. The degeneration is slowly progressive. The primary form should be scraped, since some improvement in vision may in this way be obtained.

Sclerosing Keratitis.—In certain cases of scleritis, a sector of the cornea immediately adjacent to the part of the sclera inflamed becomes intensely opaque, having undergone a fatty or hyaloid degeneration. It comes to resemble very closely the sclera in appearance, and therefore has been given the above name.

Keratomalacia is, fortunately, an extremely rare condition, and almost unknown in this country. Found in badly nourished and diseased children, it is characterised by a slow disorganisation of the cornea, as the result of extensive fatty changes in its cells. For treatment, all that can be done is careful attention to the general condition. The child usually dies from malnutrition.

CONGENITAL CONDITIONS OF THE CORNEA.

Corneal Opacities.—These are the commonest, in fact almost the only, congenital abnormality of the cornea. In origin probably non-inflammatory, they may be single or multiple, unilateral or bilateral, nebulous or intensely leucomatous. The commonest form is an intensely white marginal opacity resembling sclera in appearance. The whole cornea may be opaque, when buphthalmos or microphthalmos is usually present.

Conical cornea (keratoconus, transparent anterior staphyloma) consists in a bulging forwards of the central part of the cornea beyond its normal curvature, so that it assumes the form of an obtuse transparent cone.

The conicity may usually be seen with the naked eye by looking at the cornea from the side, but unless a careful examination is made as to the state of refraction of the eye, the early stage of this disease may be mistaken for ordinary myopia or regular myopic astigmatism. By the ophthalmometer of Javal and Schiötz (see Refraction), the reflected images are of various sizes and cannot be brought into parallel lines; this shows an irregular astigmatism of the cornea. The apex of the cone is frequently somewhat eccentric.

On throwing light into the eye whose pupil is dilated, the fundus red-reflex presents the following characteristic appearance: centrally there is a dull glow, which peripherally becomes considerably brighter, while between these two is a dark shadow of varying breadth. By retinoscopy, this shadow moves round the centre, but never across the pupil, as the mirror is rotated. If the retinoscopy mirror be so manipulated that the light is thrown *round* the cornea instead of across it, this appearance is very characteristic; it may be likened to a slowly moving catherine-wheel.

Besides this shadow, two others can usually be distinguished. In the periphery an emmetropic shadow may often be seen, though this may be replaced by the shadow of low myopia. Centrally, however, there is always present the very ill-defined shadow of high compound myopic astigmatism.

By the ophthalmoscope the vessels of the optic disc and the optic disc itself appear to be distorted, and to alter in shape

and size with each movement of the observer's head, just as occurs in looking at an object through a pane of bad glass. The first symptom of the disease is that of diminished vision, first for distant and then for near and small objects. The disease usually comes on gradually, and without pain or inflammation. As it progresses, the cornea becomes perceptibly conical in appearance and the vision sometimes so defective that the patient can read only large type (Snellen, 6, 9, or 12). Often, however, even with very great conicity, the near vision remains fairly good, but the object has to be brought extremely close to the eye. Such cases differ from simple myopia, however, in the fact that no lens improves the distant vision. In advanced cases, the top of the cone sometimes becomes opaque, but there is never perforation of the cornea, unless the case is complicated by injury.

The disease is rare, and usually bilateral, generally coming on at the age of fifteen to twenty years, sometimes later; one eye is usually attacked some little time before the other. As a rule, it undergoes a steady progress for two or three years, and then remains stationary. It appears to be more common in young women than in men, and to occur more frequently in England than in other parts of the world. It may occur in several members of the same family, and is occasionally hereditary.

The *pathology of conical cornea* is still obscure. The cornea, especially at its central part, is always thinner than normal, it being possible to push inwards the apex of the cone with a probe. It may be that this thinning of the corneal tissue is the essential feature of the affection, causing the weakened cornea to yield to the normal intra-ocular tension. On the other hand, it is possible, as was supposed by von Graefe, that the affection commences with increased intra-ocular tension, and that the thinning and bulging of the cornea are secondary to this. Against this theory, however, is the fact that no history of the symptoms which generally accompany increased tension can usually be obtained, and that the rare cases of glaucoma in subjects of this age do not follow this course. Early cases, however, are said to have been stopped by the prolonged local use of eserine or pilocarpine.

Tweedy has propounded a very feasible hypothesis. He is of opinion that, during the development of the eye, the mesoblastic material which, surrounding the optic cup, forms the fibro-vascular supports of the globe, meets last at the anterior central region—*i.e.* the centre of the cornea is the last part to be completed in the development of the outer tunics of the eyeball; and any arrest of development would materially weaken the corneal centre. He attributes the non-appearance of the cone before the age of fifteen or later to the fact that the globe has not completely finished growth, and that the tunics all yield equally until the finish, when the natural intra-ocular tension causes the weakest spot, the centre of the cornea, to bulge. Congenital defects of the iris, lens, choroid, uvula, teeth, &c., are frequently present. This fact is strongly in favour of keratoconus having a congenital origin. It is also often associated with flat-foot and spinal curvature, a fact pointing to the probability of malnutrition being an important factor in its etiology.

Treatment.—The treatment of this very serious affection has received much attention during the last half-century. Bowman, Critchett, Graefe, Donders, Wecker, and many others, have spared no pains in their endeavours to prevent its progress and to remedy its bad results.

Donders found that near vision could be improved by placing a stenopaïc disc in front of the affected eye; but the smallness of the circle which he found necessary for this purpose was too limited to be useful for distant vision, the visual field being so contracted that the patient could only see objects which are situated immediately in front of the eye. These stenopaïc spectacles are therefore only useful for near work, such as reading, needlework, and the like. They are of but little use during the progressive stage of the disease, as, owing to the increasing myopia, they would require to be changed too frequently before the eye had reached a stationary condition. An attempt should be made to correct by retinoscopy the error of refraction present along the axis passing through the centre of the cornea. This is rendered easier if observations are made through a stenopaïc disc with a central aperture of 3 to 5 mm. placed before the patient's eye. If vision is improved by the

correction, glasses should be ordered, a central 3 mm. being alone transparent. In exceptional cases, a strong concave cylindrical glass (5 D to 15 D) without the stenopaïc circle or slit is beneficial.

The advantage obtained by the stenopaïc slit induced Bowman to try to diminish the aperture of the pupil by lateral deviation and elongation. This he succeeded in doing by the operation of *iridodesis*, and the method was attended with considerable improvements as to vision, and was at one time much practised. The delicacy of the operation, however, requires great skill in its performance, and the act of incarcerating the iris in a corneal cicatrix is one which is rather to be avoided, owing to the troubles which may be thereby set up, not only in the wounded eye, but in that of the opposite side. (See Sympathetic Ophthalmitis.)

Von Graefe first suggested imitating the contraction of tissue, which occurs in the healing of perforating ulcers, by the production of an ulcer artificially. To effect this he removed the apex of the cone; the part excised was about 2 mm. or 3 mm. in diameter, and involved about two-thirds of the thickness of the cornea. For some ten to fifteen days after this excision he applied the crayon of nitrate of silver to the exposed surface, and finally allowed the surface to heal; the result was that the curvature of the cornea became reduced and the vision considerably improved. It must be admitted that the operation of removing so thin a portion from the apex of the cone is not easy to perform. The cornea is very thin, and perforation is most likely to be the result of such an attempt. The application of nitrate of silver for so many consecutive days is, again, very painful. The operation is also certain to produce a dense opacity of the central parts of the cornea, and, in all probability, will necessitate the formation of an artificial pupil.

Bowman, acting on the same principle as that of von Graefe, determined to remove a circular piece from the apex of the cone by means of a small trephine, involving the whole thickness of the cornea. He did not apply caustic to the wound, but allowed it to cicatrise.

After the cicatrix has formed, an artificial pupil is made

opposite that part of the cornea which is thought to be most desirable.

Abadie states that he has been successful in combining the operation of iridotomy with that of trephining. He first excises the circular piece of cornea as recommended by Bowman, and then introduces the blades of the iridotomy scissors through the opening thus made, and divides the iris vertically downwards.¹ This method of Bowman, with or without modifications as to the shape of the trephine, has been extensively practised, and is often attended with excellent results in the improvement of vision.

During the after-treatment the eyelids should be kept constantly closed for a week; and as it is desirable to obtain dilatation of the pupil as soon as possible, atropine ointment (F. 37) should be applied to the outside of the closed lids.

Excision of an oval piece of the cornea is an operative procedure which is sometimes adopted. Similar in principle to the preceding, it is followed by equally good results, and takes less time in healing.

Operation.—A Graefe's linear knife is made to transfix the apex of the cone, so that the point just passes through the fore part of the anterior chamber; the distance between the puncture and the counter-puncture should not exceed 4 mm. Having transfixed in this way, the knife must be made to cut its way out in a direction upwards and forwards, the eye being held steady with the fixation forceps. The lower flap of the wound is now seized with forceps, and an oval portion is cut from it with scissors; the widest part of this portion should not exceed 1 mm. The eyelids are then closed, and the case is afterwards treated as for trephining.

I have seen very good results from this method of operating both in my own practice and in that of Cowell and Anderson Critchett. The bane of the operation is its tendency to the formation of anterior synechiæ. The scar opacity is great at first, but clears up considerably in the course of three or four months. An artificial pupil is usually necessary either before or after the removal of the flap from the cornea.

The *galvano-cautery* is also employed by some surgeons to destroy the apex of the cone, with the hope of subsequent cicatricial contraction. A small flat cautery-point about 2 mm. broad is used

¹ *Maladies des Yeux*, par Ch. Abadie. Paris, 1876.

at a dull red heat. It is gently applied over the apical area of the cone, destroying the epithelium and the thin substantia propria quite down to Descemet's membrane. In doing this the conicity seems to disappear during the process, so that the curvature of the cornea appears to be nearly normal. The cauterisation should be most thorough at the centre of the cone. It is likely to cause a small perforation here, but that is of no great consequence. Anderson Critchett¹ speaks very favourably of his results by this method, especially in those cases where the apex of the cone was eccentric. Should the resulting nebula be central, an optical iridectomy will be of great assistance. There has been considerable discussion as to whether the cautery should be used until, or short of, perforation. At the present time, the majority of surgeons who use this method of treatment agree that non-perforation of the cornea is the safer procedure and produces better results.

Whatever method is adopted, no *great* improvement of vision can be expected.

In the early stages of conical cornea, before operation has been decided upon, or when the patient will not submit to operation, the general health should be supported by tonic regimen. The application of a compress of lint to the closed eyelids daily, taking each eye on alternate days, has also been advised. The use of $\frac{1}{2}$ or 1 per cent. solution of eserine dropped into the eyes three times daily, with the hope of diminishing intra-ocular tension, may also be tried. Paracentesis of the anterior chamber at intervals may also be of benefit.

TUMOURS OF THE CORNEA.

Tumours of the cornea are very rare. They occasionally occur primarily in this tissue, but usually extend from similar growths either of the ocular conjunctiva or of the interior of the eye. The chief tumours are epithelioma, sarcoma, papilloma, and implantation cyst. Tumours of the corneal limbus are described under Tumours of the Conjunctiva (p. 103).

Epithelioma of the cornea² usually invades this structure by extension from the ocular conjunctiva; it sometimes, however, appears as a small whitish or yellowish-white nodule at the sclero-corneal junction, or it may follow a pterygium.

¹ *Trans. Ophth. Soc.* vols. xii. p. 73, xviii. p. 228.

² *Ibid.* vols. xi. p. 47, xviii. p. 117.

At first it causes but slight pain or inconvenience, and may be mistaken for a phlyctenule; sooner or later it spreads and becomes painful; the surface may soften and break down. The tumour has a greater tendency to grow outwards like a papilloma than to invade the deeper structures of the globe. The conjunctival vessels become somewhat congested, and slight excess of mucus is secreted. It may eventually prevent closure of the eyelids. Its repeated recurrence after removal, and the presence of 'cell-nests,' suggest the nature of the growth.

A section presents microscopically the typical appearance of epithelioma, viz. excessive growth of epithelial tissue, in the depths of which the cells arrange themselves in concentric circles, thus assuming a 'nest-like' form.

Treatment.—Complete removal of the diseased tissue is the only way of preventing the spread of this new-growth. This may be attempted by scraping with the lupus scoop, or by excising with a knife. As a rule, the disease returns and spreads to the surrounding tissues. Under such circumstances the eye had better be enucleated, and any surrounding tissues that may be affected should at the same time be cut away.

Sarcoma of the cornea more commonly occurs by extension from neighbouring tissues. It varies in its rate of progress, but as a rule is rapidly destructive. *Treatment* consists in early excision of the eye and all surrounding tissues which may be implicated.

Papilloma of the cornea has been described, but is of extreme rarity. Under this heading may be placed, for convenience sake, a case recorded¹ by Arnold Lawson under the term 'cicatrix horn growing from the cornea.'

Epithelial Implantation Cyst.—This rare condition is the result of an injury by which some of the corneal surface epithelium has been carried into the substantia propria, and there undergoes mucoid degeneration.

Corneal Leprosy.—This rare disease may be mentioned in this place. The form of leprosy which attacks the cornea is the tuberculous variety. Perforation is very liable to take

¹ *Trans. Ophth. Soc.* vol. xx. p. 73.

place with secondary implication of the iris. Attempts should be made to arrest the progress of the disease with the galvanocautery.

DEPOSITS IN THE CORNEAL TISSUE.

Lead Deposits.—When lead lotion is used in ulcer or abrasion of the cornea, an incrustation of lead carbonate is formed in Bowman's membrane, and appears as an opaque milky-white patch. This can be removed either by scraping or by excision *en masse* of the superficial part of the cornea in which the foreign substance is lodged.

Scraping is best performed by means of a small lupus scoop. The eyelids are separated by a speculum, and the globe held in a convenient position by fixation forceps. By gentle scraping, first the epithelium is gradually removed and then the deposit. Cocaine should be used before operation. After the operation, atropine is instilled, the lids closed, and a sterilised gamgee pad applied.

Excision of the deposit is performed with a Beer's cataract-knife. An incision is made all round the deposit into the corneal tissue, and the whole superficial part of the cornea thus marked out is carefully dissected up. The after-treatment is the same as for scraping.

Calcareous deposits are occasionally found in the corneal substance of old people. These deposits have been dissolved, after the epithelium has been removed, by a 5 per cent. solution of hydrochloric acid, the excess acid being neutralised by a solution of carbonate of soda.

Bloodstaining of the Cornea.—After severe hæmorrhage into the anterior chamber, whatever the cause, the cornea is more or less completely stained a rusty colour. This is due to the deposit of hæmatoidin and hæmoisderin in fine particles throughout the substantia propria, the result of the diffusion of hæmoglobin through Descemet's membrane.

CHAPTER V.

THE SCLEROTIC.

ANATOMY AND PHYSIOLOGY — EPISCLERITIS — SCLERITIS — PERIODIC TRANSIENT EPISCLERITIS — GUMMA — TUBERCULOUS NODULE — LEPROUS NODULE—NEW-GROWTHS—CYSTS—OSSEOUS DEGENERATION.

ANATOMY AND PHYSIOLOGY.

THE sclerotic is a strong opaque fibrous structure continuous with the cornea, from which it extends backwards so as to complete the external coat of the eye. Its outer surface is white and smooth; its inner surface is of a light brown colour. It is thickest (1 mm.) at the back part of the eye, and thinnest (0·35 mm.) about 6 mm. from the cornea; at the point of union with the latter it again becomes thicker. Posteriorly it is pierced by the optic nerve at a point about 2·5 mm. internal to the antero-posterior axis of the globe. The sclerotic receives the insertions of the four recti muscles and the two obliques (see p. 550).

At the opening through which the optic nerve passes the sclerotic is not altogether absent, for it sends across fine trabeculæ, which form a sieve-like membrane through which the nerve-fibres pass. This, which is called the *lamina cribrosa*, is composed of an interlacement of bundles of white fibrous tissue. The lamina cribrosa, however, is not entirely formed from the sclerotic, since the choroidal coat gives a contribution. This is the weakest part of the ocular walls, and is the first to yield to increased intra-ocular tension.

The sclerotic is composed of bundles of white fibrous tissue, many of which are arranged either meridianly or equatorially, though a considerable number are irregularly disposed. Between these bundles lie the sclerotic corpuscles, which are placed in lymph lacunæ. Unlike what is found in the cornea, canaliculi are for the most part absent, and consequently the corpuscles are not provided with fine processes. The lamina fusca of the choroid (see p. 174)

is by some considered to be a part of the sclerotic. Elastic fibres are present in varying quantity, and a few pigment cells are usually found in the deeper layers. The texture of the sclerotic is permeated by a network of capillaries having very wide meshes. These are derived from: (a) the short posterior ciliary arteries, round the optic nerve; (b) the long posterior ciliary arteries, at a little distance in front of this; (c) the venæ vorticosæ, immediately behind the equator; and (d) the anterior ciliary arteries, close behind the sclero-corneal junction, which form a vascular ring—the ciliary or circum-corneal zone—terminating in loops at the periphery of the cornea.

The *canal of Schlemm* is a small flattened somewhat oval space, situated in the anterior part of the sclerotic, close to its junction with the cornea. Its inner wall is lined by endothelium, and it probably communicates with the fine clefts between the fibres of the ligamentum pectinatum, and so with the anterior chamber, by means of fine stomata between the individual endothelial cells. The precise manner in which it communicates with the anterior ciliary veins in its immediate vicinity is still disputed. In all probability certain valvular arrangements exist which, under ordinary conditions of intra-ocular pressure, allow the contents of the canal to pass outwards, either directly into the veins or into lymphatic spaces surrounding the latter. Occasionally the canal of Schlemm is subdivided into two or more compartments by fine trabeculæ.

The *ligamentum pectinatum* is situated just inside the sclero-corneal junction. It is intimately attached to this part, and thence extends to the iris, the ciliary processes, and the ciliary muscle. Its trabeculæ and lamellæ are composed of elastic fibres, which are derived from the splitting up of the membrane of Descemet. Between these trabeculæ are intercommunicating clefts, the *spaces of Fontana*. The epithelium from the posterior surface of Descemet's membrane is continued over these fibres, and on to the anterior surface of the iris.

Between the sclerotic and the anterior part of the ocular conjunctiva is found some loose connective tissue; this, which varies in amount in different individuals, is called episcleral tissue.

Covering the sclerotic is the *capsule of Tenon*. This is a fibrous capsule, which envelops the sclerotic and sends off processes in various directions. Anteriorly, it extends to within about 3 mm. of the cornea, and blends with the sclerotic and conjunctiva. Another portion passes in a radial direction behind the conjunctiva and the palpebral ligament, to become united with the periosteum; other reflections take place along the ocular muscles in the form of

sheaths. Posteriorly, the capsule is continued along the optic nerve as far as the optic foramen. This capsule is lined by flattened endothelial cells, similar to those of serous membrane. It forms a socket in which the globe can rotate in any direction. Its cavity communicates with the space between the lamina fusca and the lamina suprachoroidea by means of the perivascular lymph-spaces surrounding the venæ vorticæ.

EPISCLERITIS AND SCLERITIS.

Inflammation of the sclerotic is of comparatively rare occurrence, and is usually confined to the circumcorneal or anterior zone which lies between the cornea and equator of the globe. Sometimes only the superficial (episcleral) portion of the tunic is affected, whilst in more serious cases both the superficial and deeper layers are involved. The superficial form, *episcleritis*, at first appears as a dusky red nodular swelling beneath the ocular conjunctiva. One or more patches may occur; they usually occupy the portion of the sclerotic opposite to the palpebral fissure. They are not movable upon the sclerotic, but the conjunctiva can be freely moved over them. The violet tint is due to the episcleral vessels showing through the congested conjunctiva. The affection is usually very slow in progress, often lasting many months. It does not go on to suppuration, neither does ulceration ever occur. It is liable to recur, and may leave one portion of the sclerotic and crop up at another place. It mostly occurs in adults. It is often unattended by pain or other subjective symptoms; it is frequently tender; occasionally there is considerable discomfort from photophobia and lachrymation, in addition to unsightliness of the eye. The vision is unaffected.

When the deeper portions of the sclerotic are affected (*scleritis*) the swelling may be associated with *episcleritis*, but generally extends over a larger surface, frequently all round the circumcorneal zone; the swelling is, therefore, less nodular and sharply defined. The greater part or even the whole zone is of a dusky bluish-red colour. It usually attacks both eyes, and runs a very protracted course. It does

not lead to suppuration, but causes absorption or thinning of the superficial layers of the sclerotic, so that the underlying uveal tract gives the circumcorneal zone a bluish dusky aspect; sometimes the thinning is such as to lead to staphyloma of the ciliary region. This deep form of scleritis differs from episcleritis in the fact that it usually extends to the cornea and the uveal tract. If the case is at all severe, it will be found that there is haziness of the deep layers of the cornea, also signs of serous or plastic iritis and cyclitis, with the formation of posterior synechiæ and even occlusion of the pupil; the vitreous is often found to be hazy, owing probably to choroiditis as well as cyclitis. The visual acuteness is consequently impaired; pain, photophobia, and lachrymation are usually more or less severe. The disease is, therefore, one which may terminate very seriously; yet in many cases the corneal opacity disappears, the vitreous becomes clear, and other inflammatory troubles subside, leaving the vision good, although the dusky ring surrounding the cornea gives the eye a peculiar and unhealthy appearance.

Causes.—Both superficial and deep forms are found associated with rheumatism, gout, syphilis, influenza, anæmia, and dysmenorrhœa. Often, however, no other disease is apparent. Patients suffering from gonorrhœal rheumatism are sometimes attacked with scleritis, associated with mucopurulent conjunctivitis.

Treatment is of little avail either in superficial or deep scleritis. *In the superficial form* the eyes may be protected by coloured glasses. Should subjective symptoms be present, soothing remedies, cocaine, atropine, and warm fomentations may be used locally, but on no account should irritating remedies, such as zinc or cupric sulphate, be employed. Subconjunctival injection of cyanide of mercury 1 in 5,000 (see p. 135), once or twice a week, is recommended by Snellen. Pagenstecher considers that massage with yellow precipitate ointment 1 per cent. accelerates the absorption of the nodules; the ointment is introduced to the palpebral sac and rubbed in by placing the finger outside the upper lid. Iodide of potassium and salicylate of sodium, taken internally, are often useful. *In deep scleritis* the treatment must vary with the local and

general conditions. When pain, iritis, and keratitis are impending or present, leeching, warm fomentations, atropine, and cocaine are indicated; whilst the general or associated affection, such as rheumatism or syphilis, must be treated *secundum artem*.

Periodic Transient Episcleritis.—This rare disease, first described by von Graefe, and later by Fuchs, is a localised episcleritis whose characteristic is its marked tendency to recur. The ocular conjunctiva and episcleral tissues are acutely inflamed in the form of a quadrant, which often passes in a circle round the cornea, involving different quadrants in turn. No nodules are, as a rule, formed, and the attack rarely lasts more than a week. The iris and ciliary body may be involved. The cause is uncertain, but some constitutional taint is probably present, and many consider the so-called uric acid diathesis to be the main etiological factor. Others have thought the disease to be a manifestation of some vaso-motorial disturbance. Photophobia and lachrymation are marked, and considerable pain and tenderness are often complained of. Local treatment should consist of the instillation of atropine, fomentations, and complete rest; dark glasses should be worn. The patient's habits must be carefully regulated, and salicylate of sodium by the mouth often affords relief.

AFFECTIONS OF THE SCLEROTIC IN THE SPECIFIC DISEASES.

Gumma of the Sclerotic.—This rare affection may be superficial or deep. If the former, it closely resembles the nodule found in ordinary episcleritis. It occurs as a tertiary manifestation of syphilis. Treatment must be antisyphilitic.

A **tuberculous nodule** may occur in the sclerotic, though it is of great rarity.

Leprous nodules involving the sclerotic have been described.

NEW-GROWTHS OF THE SCLEROTIC.

These are all very rare. The following have been described: *fibromata*, *enchondromata*, *osteomata*, *sarcomata*, and *carcinomata*.

Melanotic spots are occasionally found in the superficial

layers of the sclerotic. They are especially common in the eyes of negroes, and are thought by some to predispose to malignant disease, as is the case with similar spots in the skin.

CYSTS OF THE SCLEROTIC.

A few cases of true cysts have been reported. They contain a clear liquid, and are placed in the superficial layers of the sclerotic.

DEGENERATION OF THE SCLEROTIC.

Osseous degeneration often occurs in old age, and in *phthisis bulbi*.

CHAPTER VI.

THE TUNICA VASCULOSA OR UVEAL TRACT.

ANATOMY — PHYSIOLOGY — HYPERÆMIA OF IRIS — IRITIS AND CYCLITIS —
OPHTHALMOSCOPIC APPEARANCE OF THE NORMAL CHOROID—HYPER-
ÆMIA OF THE CHOROID—CHOROIDITIS—IRIDO-CHOROIDITIS—TUBERCLE
OF THE CHOROID—GUMMA OF THE CHOROID—SYMPATHETIC IRRITATION
—SYMPATHETIC OPHTHALMITIS—CHOROIDAL DETACHMENT—TUMOURS—
CYSTS—CONGENITAL DEFORMITIES—OPERATIONS—IRIDECTOMY—IRIDO-
DESIS—IRIDOTOMY.

ANATOMY.

The **uveal tract**, the second tunic of the eye, is found immediately beneath the sclerotic. It consists of three parts which from before backwards are respectively called the iris, the ciliary body, and the choroid.

The **iris** is the anterior part of the tunica vasculosa, which is suspended in front of the crystalline lens. It is the beautifully coloured and contractile membrane which is seen through the transparent cornea. By its circumference it is attached to the ligamentum pectinatum and to the ciliary body. Its anterior surface is free, whilst the posterior surface rests by its pupillary edge against the capsule of the crystalline lens ; an absence of this support causes the iris to be tremulous—*iridodonesis*. It separates the anterior chamber, between it and the cornea, from the posterior chamber, between it and the lens. Slightly to the nasal side of its centre is the aperture of the pupil, whose diameter varies, when the iris is resting, from 3 mm. to 6 mm. Children, dark people, and myopes usually have larger pupils ; elderly people, fair subjects, and hypermetropes smaller. In thickness the iris is about 0·4 mm.

Developmentally, the iris consists of two parts, the anterior portion being mesodermic, the posterior portion ectodermic, in origin. The mesodermic iris is composed of the following layers :

1. The *anterior endothelium*, continuous with and similar to the

lining cells of Descemet's membrane and the ligamentum pectinatum. It consists of endothelium with large stomata or crypts, which bring into communication the aqueous chamber with lymphatic spaces and channels in the stroma of the iris.

2. The *substantia propria*, which consists of a stroma of connective tissue containing branching connective-tissue cells, many of which, in dark eyes, contain pigment-granules. The anterior part of this layer is somewhat condensed and is free from blood-vessels; this has been termed the anterior boundary layer. Within this stroma are found muscular fibres, blood-vessels, lymphatic spaces and channels, and nerves. The *muscular fibres* are of the unstriped variety, and are arranged to form two definite tracts. One of these, the *sphincter* or *constrictor pupillæ*, consists of a flattened ring around the edge of the pupil nearer to the posterior than to the anterior surface of the iris. The second muscular tract, the *dilator pupillæ*, is radially placed in the posterior part of the stroma, immediately anterior to the membrane of Bruch. It is composed of delicate spindle-cells, which cannot be seen unless the iris is first bleached. Some observers, however, deny that these fibres are muscular, looking on them rather as elastic tissue.

3. A *hyaline thin membrane* (*membrana pigmenti*), the posterior limiting membrane, or membrane of Bruch, which is continuous with the lamina vitrea of the ciliary body and choroid.

The ectodermic iris consists of :

4. The *uvea*, which is composed of two layers of pigmented cells : (a) an anterior layer of spindle-cells, the continuation of the pigmented layer of the retina; and (b) a posterior layer of polygonal cells, the representative of the remaining layers of the retina. These layers can only be seen in albino irides, or after the iris has been bleached.

The iris in health presents a brilliant appearance; its colour in dark eyes is due to the presence of pigment-granules in the connective-tissue corpuscles of the *substantia propria*, especially those in the anterior layers of the iris. If pigment is absent from the stroma, and only present in the uvea, the iris appears blue when comparatively thin, as is the case in children and young adults; where the iris becomes thicker, the colour changes to grey. The eyes of newly born children, even among the dark races, are always blue, since at this age the pigmentation is confined to the uvea. If pigment is absent from both the stroma and the uvea, the eye appears pink, owing to the choroidal reflex shining through the iris; this is seen in albinos.

The **ciliary body** is that part of the tunica vasculosa which extends backwards from the base of the iris to the ora serrata. Anatomically it consists of three parts: (1) an anterior thick portion, which supports the ciliary processes, *pars plicata* or *corona ciliaris*; (2) a posterior portion devoid of processes, which gradually thins off into the choroid, *pars non-plicata* or *orbicularis ciliaris*; and (3) the *ciliary muscle*. The ciliary processes, about seventy in number and between 2 mm. and 3 mm. long, are composed of a connective-tissue stroma, similar to that of the iris and continuous with it, and limited externally by the ciliary muscle. Lining them, as well as the *pars non plicata*, are two layers of cells separated from the underlying connective tissue by the *lamina vitrea*. The outer layer is pigmented and continuous with the pigmentary epithelium of the retina behind and with the anterior flattened cells of the uvea on the posterior surface of the iris in front. This layer of cells, from the pupillary margin in front, to the edge of the optic disc behind, is sometimes called the *uveal tract*. The inner layer devoid of pigment-granules is the sole representative of the remaining layers of the retina in front of the ora serrata, and is called the *pars ciliaris retinae*, which is continued forwards on to the back of the iris as the *pars retinalis iridis*; it consists of two kinds of cells, one set being cuboidal, granular, and with prominent nucleus; the other set being extremely fine and elongated so as to form slender fibrils which aid in the formation of the suspensory ligament. Secretory tubular glands have been described and demonstrated by Treacher Collins.¹ They exist for the greater part at the junction of the plicated and non-plicated portions of the ciliary body.

The *ciliary muscle* (Bowman) arises from the fibres of the ligamentum pectinatum opposite to the sclero-corneal junction; from this origin the greater part of its fibres (meridional) pass directly backwards to be inserted into the external layers of the choroid. The different muscular bundles are separated by small tracts of connective tissue. Other fasciculi (oblique), more loosely arranged, pass inwards to the ciliary processes; these run divergently, and frequently anastomose with one another; having reached the inner side they become circular. Others, the most internal, on reaching the base of the iris, pass into a direction almost circular, forming the circular muscle of Müller.

In hypermetropes this annular muscle is more developed than in the emmetropic eye. In myopes, on the contrary, the circular

¹ *Trans. Ophth. Soc.* vol. xi. p. 55.

fasciculi are feebly developed, the meridional fibres constituting nearly all the muscle (A. Iwanoff).

The fibres are of the unstriped variety; the muscle possesses a network of capillaries and a plexus of non-medullated nerve-fibres, with numerous ganglion cells.

(For the action of the muscle, see Refraction.)

The *iridic angle* is the space situated at the periphery of the anterior chamber between the base of the iris, which forms its posterior boundary, and the cornea in front. At the junction of these two structures are situated the fibres of the ligamentum pectinatum, between which are the spaces of Fontana leading into the canal of Schlemm.

The **choroid** is the posterior part of the tunica vasculosa, which extends from the ciliary body to the optic disc, and lies between the sclerotic externally and the retina internally. On microscopic section, it presents from without inwards the following parts:

1. The *lamina fusca*.—This consists of lamellæ of loose connective tissue containing branching pigment-cells; it adheres to the sclerotic when that is separated from the choroid, and from this fact it is sometimes described as belonging to that tunic.

2. The *lamina suprachoroidea*, which is similar in structure to the lamina fusca, being composed of lamellæ of branched pigmented connective-tissue corpuscles and a network of elastic tissue; when the choroid is separated from the sclerotic this part adheres to the former. The space between the lamina fusca and the lamina suprachoroidea is lined by endothelium and is considered to be a *lymph space*.

3. The *lamina vasculosa* consists of a dense network of large intercommunicating veins so closely connected that in some parts the intervascular spaces are less in diameter than the veins themselves. This venous plexus ends abruptly at the ora serrata. These veins are tributaries of the venæ vorticosæ. Within the fenestrations, which are chiefly fusiform in shape, are pigmented connective-tissue corpuscles, the presence of which renders this membrane uniformly dense and reflective in most eyes; a deficiency or excess of these pigment-cells will cause the outline of the choroidal vessels to be seen.

4. The *chorio-capillaris* or *membrane of Ruysch* is a reticulated vascular membrane of closely intercommunicating capillaries of large diameter; the meshes of this network are small. Here the venæ vorticosæ begin in capillary whorls, the *stars of Winslow*. There are no pigment-cells in this layer, and few, if any, round cells. A delicate structureless membrane, the *elastic layer* of

Sattler, is supposed to exist between this and the former layer. This, *Sattler* believes to be the remains of the *tapetum*, a definite layer found in the choroid of certain animals. In carnivora, the *tapetum* is composed of endothelial cells containing minute crystals, producing a shining appearance in the dark. In other animals it is fibrous in nature, and since the retinal pigment is absent from certain spots, interference of light is produced, causing a similar appearance.

5. The *lamina vitrea*, or *membrane of Bruch*, is a hyaline membrane. It is continued forwards into the ciliary body and iris. It supports the pigmentary epithelium of the retina; and just as this layer, formerly thought to belong to the choroid, has been shown to be epiblastic in origin, so the *lamina vitrea* is in all probability a layer of the retina, and not of the choroid, being produced originally from the cells of the pigment layer of the retina.

The **blood-supply of the tunica vasculosa** is very free (see fig. 46), and is divided into two distinct regions, the posterior part or choroid being supplied by the *short posterior ciliary arteries*, whilst the ciliary body and the iris are supplied by branches from the *long posterior ciliary* and the *anterior ciliary arteries*. The *short posterior ciliary arteries*, two in number, break up into eighteen or twenty branches as they are about to pierce the sclera close to the optic nerve, and pass through the *lamina fusca* into the deeper part of the *lamina suprachoroidea*; they divide dichotomously, and ultimately pass into the capillaries of the *chorio-capillaris*. Except in the region of the optic nerve, where a circular arterial anastomosis exists around the disc with small branches of the *arteria centralis retinae*, the anastomosing branches being called the *cilio-retinal arteries*, the branches do not anastomose much with one another. Anteriorly they receive a few anastomotic communications from the arteries of the ciliary region.

The *long posterior ciliary arteries*, two in number, arising directly from the ophthalmic artery, pierce the sclerotic close to the optic nerve, one on either side, and pass forwards between this and the choroid as far as the ciliary body. They give off no branches until they arrive at the ciliary region. Close to the posterior border of the ciliary muscle, each divides into two branches, which take a circular direction parallel to the equator of the eyeball, and unite with branches from the *anterior ciliary arteries* to form the *circulus iridis major*, which lies between the meridional and circular portions of the ciliary muscle.

The *anterior ciliary arteries*, eight in number, are supplied from the muscular branches of the ophthalmic artery, two from each

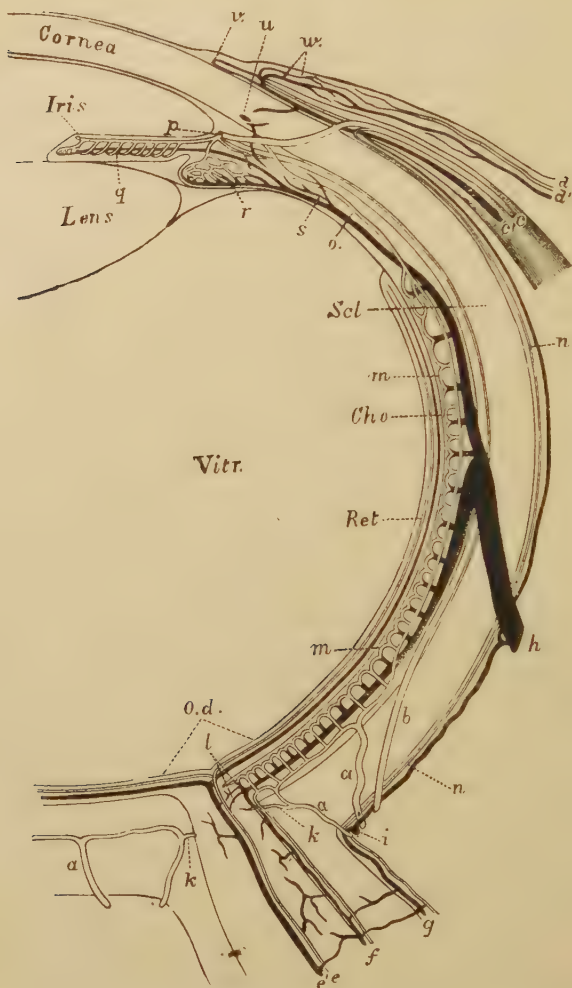


FIG. 46.—Diagrammatic Representation of the Course of the Vessels in the Eye. Horizontal Section. (After Leber.) The veins are represented black; the arteries clear.

α, arteriæ ciliares posteriores breves; *b*, arteriæ ciliares posteriores longæ; *cc'*, arteriæ et venæ ciliares anteriores; *dd'*, arteriæ et venæ conjunctivales posteriores; *ee'*, arteriæ et venæ centrales retinæ; *f*, vessels of the internal, and *g*, of the external optic sheath; *h*, vena vorticalis; *i*, venæ ciliares posteriores breves; *k*, branch of the posterior short ciliary artery to the optic nerve; *l*, anastomoses of the choroidal vessels with those of the optic nerve; *m*, chorio-capillaris; *n*, episcleral branches; *o*, arteria recurrens choroidalis; *p*, circulus arteriosus iridis major; *q*, vessels of iris; *r*, of the ciliary processes; *s*, branch to the vena vorticalis from the ciliary muscle; *t*, circulus venosus; *u*, marginal loop plexus of the cornea; *v*, arteria et vena conjunctivalis anterior; *o.d.*, optic disc.

branch to the recti muscles ; after piercing the tendon of the corresponding rectus muscle, they penetrate the sclera 2 mm. behind the sclero-corneal junction and, reaching the base of the iris, enter into the formation of the *circulus iridis major*.

The *circulus iridis major* gives off branches to the ciliary processes, which divide up into numberless fine branches. It also gives branches to the iris, which pass radially towards the pupillary margin, close to which they form an anastomotic ring, the *circulus iridis minor*, from which, in the foetal eye, fine branches pass to the pupillary membrane.

The iridic arteries have thick middle and outer coats, and but little muscular tissue. The *veins* of the tunica vasculosa are somewhat different in their mode of termination from that of the arteries. Thus the anterior ciliary veins are quite small, and receive blood only from part of the ciliary muscle. They anastomose with the conjunctival veins and with Schlemm's canal. The blood from the veins of the iris and from the rest of the ciliary body is returned to the choroidal veins.

The veins of the choroid anastomose very freely with one another ; they do not accompany the posterior short ciliary arteries, but are arranged in curves (*venæ vorticosæ*) as they converge to four or five principal trunks ; these pierce the sclerotic very obliquely about halfway between the optic nerve and the cornea to join the ophthalmic vein (*h*, fig. 46).

The **nerve-supply of the tunica vasculosa** is derived from the long ciliary branches of the nasal branch of the first division of the fifth nerve, and from the short ciliary nerves, about fifteen in number, branches of the lenticular ganglion, being derived from the oculomotor nerve. Piercing the sclerotic near the optic nerve entrance, they pass forwards between this tunic and the choroid, lying in grooves on the deeper surface of the former. A plexus is formed in the lamina suprachoroidea, in the meshes of which ganglion cells are found ; these nerve-filaments are chiefly vasomotorial in action. On reaching the ciliary bodies, the ciliary nerves form a plexus, from which fibres pass to the ciliary muscle. Passing on, a second plexus is formed in the iris, from which vasomotorial branches supply the vessels ; muscular branches from the third nerve supply the constrictor pupillæ ; sympathetic fibres pass to the dilator pupillæ ; while sensory fibres from the fifth nerve supply the anterior surface of the iris. Ganglion cells are found in the ciliary body, but not in the iris.

The Lymphatics of the Tunica Vasculosa.—In the iris there are no distinct lymphatic vessels, but the sheaths of the blood-vessels contain lymphatic sinuses, and the whole stroma is riddled with lymph

spaces. These open into the aqueous chamber, and also into the spaces between the fibres of the ligamentum pectinatum, and so reach the canal of Schlemm.

Between the ciliary muscle and the sclerotic is a lymph space, limited anteriorly by the attachment of the ciliary muscle. It is a common seat of inflammatory exudations or hæmorrhage in cyclitic disease. This space is continuous with a similar one in the choroid, between the lamina fusca and the lamina suprachoroidea, which communicates, by means of perivascular sheaths surrounding the venæ vorticosæ, with the lymph space within the capsule of Tenon.

The Lymphatics of the Eye.—Schwalbe¹ has shown that there exist in the eye several spaces in which lymph is formed, and from which it is discharged in three directions. These he classifies into an anterior and two posterior systems.

The *anterior lymphatic system* comprises the canal of Petit, the aqueous chamber, the spaces of Fontana, the canal of Schlemm, and the venous or lymphatic plexus in connection with this canal. The lymph secreted by the ciliary processes travels to the aqueous chamber by three channels; a large proportion passes to the vitreous humour and the canal of Petit, and thence through the suspensory ligament to the aqueous chamber, then forwards through the pupil; another portion passes directly into the aqueous chamber, and then forwards through the pupil; a third current takes place from the ciliary processes through the base of the iris into the periphery of the aqueous chamber. The *aqueous humour* thus formed leaves the aqueous chamber at the angle between the iris and the cornea by passing through the meshwork of the ligamentum pectinatum (spaces of Fontana); it then reaches the canal of Schlemm, where there exists a system of valves through which the aqueous passes directly into the plexus of veins in its immediate vicinity. Having thus reached the blood-current, it is conveyed to the choroidal veins.

The *posterior lymphatic spaces* are two in number—viz. those of the choroid and the sclerotic, and those of the retina and optic nerve. The former of these has already been mentioned as existing between the lamina fusca and lamina suprachoroidea; this space communicates, by means of perivascular sheaths surrounding the venæ vorticosæ, with the lymph space within the capsule of Tenon, which, as we have seen, extends along the outside of the optic nerve, through the cranium, and into the lymphatics of the neck.

The lymphatics of the retina form sheaths to the blood-vessels, and so pass to the optic nerve. The optic nerve also possesses

¹ Stricker's *Handbook of Histology*.

another lymph space between its pial and dural sheaths, *the inter-sheath space*, which communicates posteriorly with the subdural cavity, and terminates anteriorly at the lamina cribrosa.

PHYSIOLOGY OF THE TUNICA VASCULOSA.

Movements of the Pupil.—The iris, with its central aperture, serves as a diaphragm to shut off marginal rays of light, and thus prevents spherical aberration. It also regulates the amount of light entering the eye. The size of the pupil depends upon the state of contraction of the two antagonistic sets of muscular fibres, the sphincter and the dilator pupillæ.

Contraction of the pupil to light is brought about by the action of the sphincter pupillæ, which is governed by a reflex mechanism, of which the optic nerve is the afferent path and the oculo-motor nerve the efferent path. The nucleus of origin of the third nerve is situated in the fore part of the floor of the aqueduct of Sylvius, and it is the middle portion of this which is the centre for the contraction of the pupil, the anterior part governing accommodation, the posterior and greater portion the action of the extra-ocular muscles. The fibres for the intra-ocular muscles leave the lower division of the main trunk by the short root to the lenticular (ophthalmic) ganglion, and enter the globe by means of the short ciliary nerves. The method for examining the pupillary light reflexes is as follows : Cover both eyes with the palms of the hands ; upon exposing one eye—for example, the right—to the light, its pupil contracts (*direct light reflex*), and the left pupil also contracts, though to a less extent (*indirect or consensual light reflex*), being indirectly stimulated by the light entering the right eye ; upon exposing the left eye, a further slight contraction occurs in the right eye (consensual reflex) in conjunction with the direct reflex in the left. This consensual light reflex cannot be obtained in those animals whose optic nerves decussate completely. If, after shading the eyes for a few minutes, they are suddenly exposed to a bright light, a slight and temporary rhythmical contraction and dilatation of the pupils ensue—*hippus* ; this is very evident in neurotic individuals. Contraction of the pupil also occurs : (1) When the optic nerve is stimulated by other agents, as electricity. (2) When the eyes are accommodated for near vision. (3) In the early stages of poisoning by chloroform, alcohol, &c. ; and in nearly all stages of poisoning by morphia, eserine, and some other drugs. (4) In deep sleep. (5) After the local application of eserine and other miotics. (6) When the middle part of the third nerve nucleus, or the nerve-trunk prior to the exit of the short root to the

lenticular ganglion, is irritated. (7) With a destructive lesion of the cervical sympathetic, or of the lower cervical part of the spinal cord.

Dilatation of the pupil is brought about by the dilator pupillæ, which is governed by a reflex mechanism, just as is the constrictor pupillæ. The dilator pupillæ, as to the existence of which there has been much controversy, has been demonstrated both physiologically and anatomically. It is supplied by fibres of the sympathetic nerve, which have a nucleus in the spinal cord about the region of origin of the first and second dorsal nerves, the cilio-spinal centre of Budge and Waller, subordinate to a higher nerve-centre in the medulla. The fibres leave the cord by the lower cervical and upper dorsal anterior nerve-roots, and, passing into the last cervical and first thoracic ganglia, enter the sympathetic cord and are conducted upwards to the Gasserian ganglion, and through the nasal nerve enter by the long root the lenticular ganglion, from which they proceed to the eyeball. Dilatation of the pupil occurs: (1) When the stimulus of light is withdrawn from the retina, as by passing from a bright into a dim light. (2) When the eye is adjusted for distant vision. (3) In the later stages of poisoning by chloroform, and in all stages of poisoning by atropine and certain other drugs. (4) After the local action of atropine and other mydriatics. (5) With a destructive lesion of the middle part of the third nerve nucleus, or of the nerve-trunk prior to the exit of the short root to the lenticular ganglion. (6) When the cervical sympathetic or the lower cervical part of the spinal cord is irritated. (7) Reflex dilatation of the pupil may be produced by cutaneous irritation, such as an electrical stimulus, or a pinch or prick of the skin; this is particularly evident when a patient is under complete anæsthesia from chloroform. In this category also come the dilatation caused by mental emotion, as fear, anxiety, and the like, and that produced during dyspnoea and other violent muscular efforts.

The Mechanism of Accommodation.—See p. 476.

The Function of the Ciliary Processes.—The ciliary processes have a double function: they support the lens by means of the suspensory ligament, which is partly attached to them; and they take part in the production of the aqueous humour. Exactly what portion of their structure is engaged in this secretion is still a matter of some doubt. The ciliary glands, discovered by Treacher Collins, are considered by him to be the producer of the fluid, but it is doubtful whether they are sufficiently numerous, and it seems better to consider that the processes as a whole are the structures concerned. It has been suggested that the function of these glands is the formation of pigment. They are found in greater number in dark eyes than in blue eyes, and appear to be absent in albinos.

The ciliary processes, by their proximity to the edge of the lens, are considered to be the chief agents of nutrition to that body (Brailley).

The Function of the Choroid.—In the first place, the slight anastomosis between the vessels of the choroid at the edge of the optic disc and those of the optic nerve at the same place has some influence in the nutrition of the optic nerve and retina. Secondly, the capillary layers of the choroid are of great importance in the general nutrition of the eye, and in the regulation of the intra-ocular tension. In addition, the chorio-capillaris also affords nutrition to the outer layers of the retina, and also, in conjunction with the ciliary processes, to the vitreous.

HYPERÆMIA OF THE IRIS.

Hyperæmia of the iris attends many local inflammatory changes, as purulent ophthalmia, acute catarrhal conjunctivitis, and other severe forms of conjunctival inflammation; keratitis, especially if due to a foreign body deeply embedded in the substance of the cornea; other forms of injury; also after intra-ocular operations, as iridectomy, cataract extraction, &c.; and since it is the first stage of inflammation, its early recognition, combined with prompt treatment, will often cut short an impending attack of acute plastic iritis. It is characterised by contraction and sluggish action of the pupil, slight discoloration of the iris with indistinctness of its reticulum, and engorgement of the iridic vessels, which can often be seen by a strong magnifying lens; the outline of the pupil is clear, the media are not hazy, and the pupil dilates fully and regularly under a mydriatic. There is always injection of the circumcorneal zone, and lachrymation with photophobia is present. The *treatment* mainly consists in local depletion by leeches and the instillation of atropine.

IRITIS AND CYCLITIS.

Inflammatory conditions of the iris and ciliary body are frequently found together, and in consequence a description of the general symptoms of each will precede the consideration of their etiological factors, and the influence these factors have on those symptoms.

Symptoms of Iritis.—The earliest symptoms are those of hyperæmia of the iris: 1. *The mobility of the iris is diminished.*

In all cases of inflammation of the iris, the pupil will be found to move less actively than in health. In some cases its movements are sluggish, in others completely lost. This is due to spasmodic action of the sphincter pupillæ.

2. *The colour of the iris is altered.* This change is sometimes very slight, and liable to escape notice, but by a careful examination with oblique focal illumination, there will nearly always be found a change in the colour of the tissue surrounding the edge of the pupil. In many cases this is very marked, the blue or grey iris becoming of a yellowish-green tint, whilst the dark brown colour assumes a brownish-red, or rust colour. Besides this, there is generally a dull muddy appearance of the tissue of the iris. The colour of the two irides should be carefully compared.

3. *The blood-vessels immediately surrounding the cornea are injected.* These are always seen as a pink, or deep red ring, whenever iritis is present (see fig. 8, opposite p. 118). There are also photophobia and lachrymation, with conjunctival injection.

In addition to these symptoms of hyperæmia, we have those resulting from inflammatory exudation. These include : 4. *Turbidity of the iris*, due to exudation in the iris itself, and into the aqueous fluid. This also produces a muddy appearance of the pupil. Lymph may collect in the anterior chamber (*hypopyon*), or an iridic vessel may rupture with the formation of *hyphæma*. If the lymph which occupies the pupil becomes organised, we get an opaque persistent membrane, which may be perforated by one or more small apertures. This condition is termed *occlusion of the pupil*. Again, lymph in the posterior chamber may become organised, forming adhesions between the iris and the capsule of the lens (*posterior synechia*). If the whole of the pupillary margin is adherent to the lens capsule, the condition is termed annular posterior synechia; the anterior chamber is cut off from the posterior chamber (*seclusion* or *exclusion* of the pupil). Lastly, the whole of the posterior surface of the iris may be adherent to the capsule of the lens, a condition known as *total posterior synechia*. This, however, is rarely found in absence of cyclitis.

5. *Impaired vision.* This varies very considerably in extent. Even in the slight forms, the sight will be subnormal. With occlusion of the pupil, visual acuity is very greatly diminished, perception of light alone remaining.

6. *Pain* may be entirely absent, or may exist in various degrees of severity within the eye, and in the surrounding temporal, frontal, and malar regions. The amount, however, depends largely on the etiological factor.

Symptoms of Cyclitis.—This affection—which, when it occurs without iritis, is usually chronic with few inflammatory symptoms—is recognised by (1) a *sluggish, somewhat dilated, pupil*; (2) a *deepened anterior chamber*; (3) a normal-looking iris; and (4) the presence of so-called *keratitis punctata*. Upon examining the cornea by the oblique focal illumination, or with the ophthalmoscope, small dots of opacity are seen on the back of that membrane—*keratitis punctata*. These dots may be irregularly scattered, or, as is more often the case, may occupy a triangular area, the apex of which is opposite the pupil and the base at the periphery of the cornea, either below or at one side (fig. 5, opposite p. 118). They are better named *precipitates*.

Cyclitis, when it accompanies iritis, is much more acute, and is recognised by the following additional symptoms: (5) tenderness over the ciliary region; (6) increased intra-ocular tension; (7) vitreous opacities; (8) total posterior synechia; and (9) cellulitis of the eyelids and cheek. It is rare for cyclitis to occur without iritis, the irido-cyclitis usually found being that disease formerly known as serous iritis, in contradistinction to plastic iritis with its marked exudation, as described above.

Etiology of Iritis and Cyclitis.—In considering the etiology, pathology, diagnosis, prognosis, and treatment, iritis and cyclitis will be taken together.

In a large majority of cases, the cause is some constitutional taint. It is probably only in the case of a perforating injury that iritis is a local affection. Cases of iritis, however, occur, the cause of which cannot be discovered; these, until they are better understood, must be termed idiopathic. Iritis and cyclitis may, however, be secondary to inflammation of a

neighbouring structure. Hence we have the following etiological classification of iritis and cyclitis: 1. Symptomatic; 2. Traumatic; 3. Idiopathic; and 4. Secondary.

1. *Symptomatic iritis* occurs as a result of syphilis, rheumatism, gonorrhœa, tuberculosis, gout, diabetes, or it may follow one of the exanthemata.

(a) *Syphilitic iritis*, much the commonest form of this class of iritis, comes on as a secondary symptom, as a rule within nine months after the acquirement of the initial lesion, generally appearing a few weeks after the occurrence of the rash in the fourth or fifth month. It is exceedingly rare after the second year. It occurs in about 4 per cent. of all cases of syphilis. There are four chief forms—*plastic iritis*, *iritis gummosa* or *papulosa*, *gumma* of the iris and ciliary body, and *irido-cyclitis*. The plastic form differs from the other forms in the large amount of exudation, and in the rapidity with which this becomes organised, causing marked change in the colour of the iris, especially around the edge of the pupil, and extensive adhesions. It also shows little or no tendency to recur when once it is cured, and pain and conjunctival injection are often comparatively insignificant.

Iritis *gummosa* or *papulosa* appears as a secondary manifestation of syphilis, and must not be confounded with the next form. It is a variety of plastic iritis, and occurs in 18 per cent. of cases of this form. Its characteristic feature is the formation of multiple orange- or rust-coloured nodules of lymph either at the ciliary or the pupillary margin of the iris. The ordinary signs of inflammation of the iris are present. As a result of this form, a broad posterior synechia is very common.

True *gummata* of the iris and ciliary body may occur as a tertiary symptom of syphilis, but are very rare. They usually start from the iridic angle, and cause a marked ciliary staphyloma, over which the sclerotic coat is thinned out, and in consequence has a bluish tint. Pain is frequently well marked, and vision usually greatly and permanently affected. A case, however, has been recorded¹ in which, after the correction of the resulting astigmatism, useful vision was restored.

¹ *Ophthalmic Review*, vol. xvii. p. 238.

Irido-cyclitis is a late secondary or tertiary manifestation, since it may occur several years after the primary affection. It is frequently accompanied by choroiditis.

Hereditary syphilis is occasionally characterised by *congenital syphilitic iritis*, either intra-uterine or occurring at or a few hours after birth. A little later, usually between the ages of six weeks and sixteen months, iritis may develop—*infantile iritis*. Still later, interstitial keratitis may be complicated by iritis, extensive adhesions sometimes resulting. Occasionally cyclitis with no keratitis has congenital syphilis as its cause.

(b) *Rheumatic iritis* may follow acute rheumatism, but is more common in the chronic form. It is attended with greater pain and conjunctival injection than the syphilitic form. The plastic exudation, however, is not so marked; there is consequently less change of colour in the iris, and the adhesions are fewer and form more slowly. It shows great tendency to recur, the relapses often alternating with the attacks of rheumatism, though in some cases the two are coincident. Sight may be little affected, even after repeated attacks. Both the iris and ciliary body are usually involved.

(c) *Gonorrhæal iritis* resembles rheumatic iritis very closely. It is a sequela of gonorrhœa, and occurs especially after gonorrhœal rheumatism. It may occur several years after the urethritis. Like rheumatic iritis, this form shows an especial tendency to recurrence.

(d) *Tuberculous irido-cyclitis*.—A form of iritis which is essentially chronic, and which differs chiefly in this respect from all other varieties, is occasionally seen, though from the rarity of its occurrence its earlier stages are liable to be overlooked. This disease is probably always secondary to a primary tuberculous focus elsewhere in the body, though the primary lesion may not be discovered. In the disseminated variety, the pupil is usually contracted and immobile. At first nodulated on its surface, the iris eventually fills up the anterior chamber. Its colour is a dull brick-red mottled with grey, and it appears to be very vascular. The pain and circumcorneal injection are not marked at first, though in the later stages, owing to the supervention of secondary glaucoma, the

pain becomes intense and persistent, in spite of all palliative measures; chemosis and intense circumcorneal injection accompany this increase of tension. The disease usually occurs in young subjects. The solitary form resembles very closely the disseminated variety, but starts singly.

(e) Other constitutional diseases which predispose to iritis are gout, in rare cases diabetes, and occasionally it occurs during an attack of one of the exanthemata, the commonest of which being relapsing fever.

2. *Traumatic iritis* results from perforating injuries, mechanical violence, or chemical irritation. Of these, the first is much the most common. The iritis may go on to a suppurative form, when it is characterised by marked and rapid changes in the iris. The pupil is contracted, and either sluggish or immovable. The tissue of the iris is swollen, and its colour changed to a muddy green or brownish-yellow. The aqueous humour is at first slightly turbid, but before long there is a collection of yellowish puro-lymph at the bottom of the anterior chamber, which may increase so much as to occupy the greater part of that cavity. Suppurative iritis is seldom confined to the tissue of the iris, but usually is either derived from or extends to the surrounding tissues, as the cornea, the ciliary body, the choroid, and the vitreous (see Panophthalmitis). A previous low state of health is a powerful predisposing cause. A common and typical example may be cited of a mother, suckling her child twelve, fifteen, or more months old, receiving a scratch on the cornea from the child's finger-nail. A septic ulcer of the cornea ensues, followed by suppurative iritis, panophthalmitis, and complete loss of the eyeball. Operations on the eyeball are sometimes followed by iritis, especially cataract extraction and iridectomy. Aphakic eyes appear to be especially prone to iritis, which usually takes the form of a chronic recurring inflammation. Eyes with retinal detachment are also peculiarly susceptible to iritis.

Sympathetic irido-cyclitis will be dealt with under a special heading.

3. *Idiopathic irido-cyclitis*.—A few cases of irido-cyclitis seem to have no definite cause. The iritis usually predominates. It apparently comes on spontaneously, and resembles

in every respect an acute plastic iritis, or a more chronic irido-cyclitis.

4. *Secondary irido-cyclitis* is uncommon. It usually follows a suppurating ulcer of the cornea, and more rarely is secondary to choroiditis. Under this heading may be included the form of irido-cyclitis which occurs in metastatic panophthalmitis; the causes of this disease are puerperal fever, all forms of pyæmia, and ulcerative endocarditis. The iritis is acute, suppurative, and hypopyon is usually present. The attack in one eye is, as a rule, rapidly followed by a similar attack in the other, with complete loss of vision in both.

Pathology of Iritis and Cyclitis.—In *plastic iritis* there is engorgement of the vessels, with small-celled infiltration of the whole thickness of the stroma of the iris. Exudation follows, consisting of fibrin with a varying number of leucocytes and a few red blood-corpuscles. This exudation may become completely absorbed, or it may coagulate and fall to the lower part of the anterior chamber, forming the so-called hypopyon; or it may be organised into connective tissue, synechiæ being the result.

In *cyclitis* a similar set of changes takes place, but added to them we have *keratitis punctata* or *precipitates*. Microscopically, there are found to be small aggregations of round cells cemented together by fibrin containing a small amount of pigment. They are not formed, as was thought at one time, by any inflammatory condition of the cornea, but result from an inflamed ciliary body, and are carried by the lymph stream through the pupil to the posterior surface of the cornea, on which they are deposited.

An eye affected with *tuberculous irido-cyclitis* presents, on section, a very characteristic appearance. The whole of the anterior chamber is filled by the iris, which moulds itself to the shape of the cavity, becoming adherent to the cornea in front and the lens capsule behind. It presents to the naked eye the appearance of small white nodules, massed together and interrupted here and there by the disturbed uveal pigment, giving the growth the resemblance of marble, just as is seen in a tuberculous lung. Microscopically the nodules present the characteristic features of miliary tubercle, central

giant-cells, epithelioid cells, and small round cells. They are non-vascular, though the vessels between them are enlarged and numerous. The ciliary body is usually involved, and the uveal pigment is so interrupted that it would be difficult to believe, from the appearance, that it was ever a continuous tract. The presence of the tubercle bacillus has in many cases been demonstrated.

The complications and sequelæ of iritis are not many, though important. *Synechiæ*, anterior and posterior, may occur: the former are almost invariably the result of a perforating wound or ulcer of the cornea—in rare instances they may be due to tubercular or gummatous deposits on the front of the iris; the latter (posterior synechiæ) may arise from not dilating the pupil early in an attack of plastic iritis: exclusion and occlusion are the more serious forms. As a result of annular posterior synechia, atrophy of the iris occurs, with a bulging forwards of the ciliary portion, a condition known as *iris bombé*. This closes the iridic angle, and secondary glaucoma is frequently set up. Extension of inflammation, *cyclitis* or *choroiditis*, may supervene. In suppurative iritis, *cyclitis* and *choroiditis*, as a rule, exist; in plastic iritis they are exceptional complications. Another complication is opacity of the lens, resulting from deficient nutrition; this may occur with or without *phthisis bulbi*, which is found as a sequela of suppurative iritis following perforation, and in some cases of irido-cyclitis where there has been much exudation into the posterior chamber and upon the posterior surface of the lens. Again, the inflammation may lapse into *chronic iritis*; and lastly, especially in rheumatic and gonorrhœal iritis, the inflammation may recur, each attack leading to fresh adhesions.

Diagnosis of Iritis and Cyclitis.—The more acute forms of conjunctivitis may be mistaken for an early attack of iritis. To diagnose the two diseases, special attention must be paid to the colour of the iris, and to the activity, size, and shape of the pupil. In iritis there is always some discoloration of the iris, which is especially marked at the pupillary margin; the movements of the iris are sluggish, even if not lost, the pupillary light reflex being diminished; the pupil itself is

somewhat contracted, and may be slightly irregular in shape. In all cases the two eyes should be carefully compared, and the action of atropine on the iris of the suspected eye investigated. In this way a small posterior synechia may be discovered, and the diagnosis of iritis made certain.

The acute and subacute forms of glaucoma and iritis have many signs in common, especially if cyclitis be also present. These are, conjunctival and circumcorneal injection, a muddy appearance of the iris, a fixed pupil, and increased tension. The history of the case must be carefully investigated. The onset of glaucoma is much more sudden, and is preceded by definite premonitory symptoms. During the attack the cornea becomes steamy and anæsthetic; the pupil is dilated, whereas in iritis it is usually contracted; the anterior chamber is shallow; the intra-ocular tension is raised to a greater extent than in irido-cyclitis; if the media are sufficiently clear for an ophthalmoscopic examination, the optic disc in glaucoma will be found to be cupped.

Cyclitis and episcleritis often resemble each other very closely. A circumcorneal redness is present in both, but the accompanying characteristic signs of cyclitis are usually sufficient to establish its diagnosis, while the episcleral inflammation is often localised.

The diagnosis between iritis alone and iritis accompanied by cyclitis has already been referred to.

The diagnosis of tuberculous irido-cyclitis is determined by the chronicity of the affection, by the characteristic appearance of the iris, by the presence of tuberculous disease elsewhere in the body, and by the resistance to all ordinary treatment for iritis.

The prognosis of iritis and cyclitis, if seen early, is, in the majority of instances, good. Syphilitic iritis almost invariably subsides under judicious treatment without much, if any, deterioration in vision. Rheumatic iritis is less amenable to treatment, and liable to recur; in fact, this is its chief characteristic feature. If the pupil responds to atropine completely, a good prognosis may be made. Extensive synechiæ, especially exclusion or occlusion of the pupil, are grave conditions, and liable to be complicated with secondary glaucoma;

the latter (occlusion) is attended with complete loss of vision save perception of light. Operative measures in such cases are alone attended with any success; the result may be very decided, or, on the other hand, if the condition has existed for years, much improvement cannot be entertained. Should the tension be subnormal, operative treatment is contra-indicated. The more chronic the iritis, the more likely are atrophic changes in the eyeball to occur, and so prognosis is bad. Serous irido-cyclitis, if not due to sympathetic trouble, subsides fairly readily, and with favourable results, though unrelieved tension may be the cause of glaucoma cupping, with atrophy of the optic disc, attended with partial or complete loss of sight. Sympathetic irido-cyclitis and tubercular iritis are most serious; in the former, little sight can be hoped for; recovery has occasionally taken place from the latter, with gradual absorption of the growths, but as a rule the prognosis is bad, the eye being lost, and the patient usually dying from general tuberculosis or meningitis. Suppurative iritis rarely exists as a local affection, and consequently the prognosis in these cases is also unfavourable.

Treatment of Iritis and Cyclitis.—The treatment of iritis depends to a certain extent upon the cause and kind to be dealt with. There are, however, certain important points in all cases which require strict attention; they are as follows:

1. The dilatation of the pupil.
2. The relief of pain and congestion.
3. The treatment of any constitutional dyscrasia, and attention to the general health.
4. The diminution of any increase in the intra-ocular tension.
5. The treatment of complications and sequelæ.

1. *Dilatation of the pupil* is of paramount importance, and should be done immediately the condition is recognised. The object of this is to prevent the formation of plastic adhesions to the lens or to break them down if recent, to keep the inflamed iris at rest by paralysing the sphincter pupillæ, and, by contracting the iridic blood-vessels, to diminish the amount of plastic exudation. The best mydriatic is un-

doubtedly **atropine sulphate**: it is a vaso-constrictor; it paralyses the sphincter pupillæ and ciliary muscle; it is an anodyne. It should be used in the form of drops ($\frac{1}{2}$ –1 per cent. solution) instilled into the palpebral sac every three or four hours, or as an ointment (F. 37) introduced between the lids. This causes the widest possible dilatation of the pupil, and, by keeping the pupillary edge of the iris away from the capsule of the lens, prevents the formation of posterior synechiæ. If adhesions have already formed and are recent, it is a good plan to use atropine every hour for six consecutive hours: this treatment is likely to break them down, leaving perhaps a few dots, or a ring of pigmented lymph upon the capsule, which, however, may partially or entirely disappear. Dots of pigment seen on the capsule of the lens are always indicative of previous iritis. Adhesions which a 1 per cent. solution of atropine sulphate fails to break down may be broken down by a 2 per cent. ointment used occasionally and with care. If the adhesions are of sufficient age to have become firmly organised, the atropine will not break them down; but it will still cause dilatation of any part of the pupil that may be unattached, and so prevent further complication of this kind. When once the pupil has been well dilated, and when any adhesion resists all treatment, the amount and frequency of atropine administered should be diminished, a 0·5 per cent. preparation being used three times a day.

In using atropine, it must be remembered that the ciliary muscle is temporarily paralysed, and that near vision is therefore greatly impaired for the time. Again, the use of this drug occasionally gives rise to what is called *atropine irritation* or even *atropine poisoning*. The former consists of irritable conjunctivitis, and of swelling and erythema of the skin of the eyelids and surrounding region. In some cases it is very severe. Sometimes a single application of atropine is sufficient to set up violent pain in the eyes, with photophobia, intense injection of the conjunctiva, with chemosis, great redness, swelling, and a vesicular eruption of the skin of the eyelids, cheeks, and forehead. When this complication arises, the atropine must at once be stopped, and some other mydriatic substituted. For this purpose a 0·5 per cent. solution of duboisin, or a 5 per cent. solution of hyoscyamine, or a 0·2 per cent.

solution of scopolamine (formerly known as hyoscine), should be tried with caution. Care should also be taken to compress the lachrymal sac with the finger for a minute or two after the instillation. Patients who cannot tolerate atropine are in some cases also unable to withstand the action of these agents, although as a rule these are less irritating than atropine. Atropine poisoning is recognised by heat and dryness in the mouth and throat, difficulty in swallowing, thirst, and loss of appetite. The face becomes hot and flushed, the pulse quickened, and the patient has a strange noisy manner; he may be delirious or have convulsions or hallucinations. Should such symptoms present themselves, the atropine should be stopped at once, hot coffee administered, together with a hypodermic injection of pilocarpine (half a grain) or morphia (quarter of a grain).

Besides atropinism, there are other contra-indications for the use of atropine and other mydriatics. In cases of iridocyclitis, not only is the tension somewhat raised, but the instillation of atropine may be followed by increased pain, photophobia, and lachrymation. In these cases the atropine preparation must be either very weak (about one-twelfth of a grain to the ounce) or replaced by a weak solution of physostigmine (about one-quarter of a grain to the ounce).

2. *The relief of pain and congestion.*—One of the best methods we possess of doing this is by the alternate application of moist and dry heat. For this purpose, let the eye be bathed every few hours with hot water, and then apply a large pad of hot dry cotton-wool to the closed eyelids and keep it there until the next fomentation. The wool is easily made hot by contact with the outside of a can of boiling water. The dry hot wool alone is also very comforting and beneficial. Poppy-head or belladonna fomentations (F. 24) should be applied in all acute cases, especially if the iris be suppurative, and changed every thirty or sixty minutes. Some patients prefer cold to heat, and this is best applied by a modification of Leiter's tubes, through which runs iced water. The atropine which has been used for dilating the pupil is also a sedative, and will help to relieve the pain. The eye should be shaded from the light, and on no account should caustic applications be applied to the conjunctiva or powerful astringent lotions, as sulphate of zinc, used. Cocaine can be suitably combined with

atropine; it further assists the sedative effect of the latter drug; it is a vaso-constrictor and a mydriatic; the latter action, however, is insignificant compared with atropine. The use of several leeches applied to the temple, eyelids, or malar eminence, or the application of Heurteloup's artificial leech to the temple, often gives relief. Each leech will remove rather more than one drachm of blood. Bleeding may be encouraged for an hour after the leeches have dropped off, by means of a fomentation. Should there be any difficulty in stopping the hæmorrhage, the leech-bites must be touched with lunar caustic and compressed with a pad of lint and a bandage. It is often seen that after the application of leeches the pupil responds better to atropine.

Heurteloup's artificial leech consists of a sharp cylindrical drill, and a glass exhausting-tube with an air-tight piston (see fig. 47). The drill can be set at any depth by means of a screw. It is applied to the temple, the hair having been previously shaved off from a space sufficiently large to accommodate the end of the cylinder. The blade, being set at a depth sufficient to penetrate the skin, is firmly applied to the temple, and the incision made by rotating the upper knob; this done, and the cutter being withdrawn, the exhaustion is effected by gradually rotating the screw. The cylinder holds about an ounce of blood, and ought to fill in the course of five minutes. Light should be excluded from the eye for about twelve hours after the use of the artificial leech.

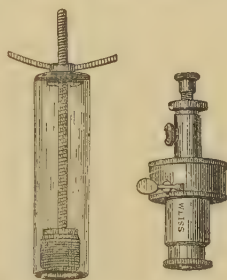


FIG. 47.—Artificial Leech.

In lieu of ordinary leeches or the artificial one, wet cupping may be performed over the temple by making a few small cutaneous incisions and applying a small glass cup from which the air has been exhausted in the usual manner.

Another important aid in the relief of pain and congestion is the operation of *paracentesis of the anterior chamber* (see p. 134). This simple proceeding will often give immediate relief when other methods are only partially successful. It is never attended by bad results, and its value should not be overlooked, especially when other means have failed, and the aqueous humour is turbid or contains puro-lymph.

In iritis resulting from injury, when seen in the early stage, the continuous application of cold to the closed eyelids, by means of lint dipped in iced water, is the best means of allaying inflammation; this should be combined with atropine and the use of leeches.

3. *General treatment* is also of great importance. It is advisable in all cases of acute plastic iritis to give a brisk purge at the onset. Calomel with colocynth and hyoseyamus as a pill, or the first drug as a powder, should be given at night, followed by a saline purge in the morning. Sleep is most essential: it gives rest to the eye as well as relief from pain. It may be procured by administering at night a bromide of potassium and chloral hydrate draught, or morphia by the mouth or hypodermically. Plethoric subjects, besides requiring leeches and the initial purge, need a daily dose of mercury—two grains of blue pill every night, followed by a slight saline draught each morning for three or four days. Diaphoresis by hot-air baths, Turkish baths, or by drugs such as Dover's powder, &c., is exceedingly valuable in treating these patients; especially if the inflammation be due to gonorrhœal or simple chronic rheumatism. Depletory treatment is contra-indicated in anæmic and weakly individuals, and in those suffering from suppurative iritis; leeches should not be used; and after the first purge, tonics, as bark and ammonia, good fresh air, and a supporting and nourishing diet, are to be recommended.

When *syphilis* is found to be the cause of the iritis, the general disease must be rigorously treated by means of mercury in addition to the local affection, but, before a course of this drug is begun, certain precautions must be taken. It is necessary that the mouth should be in a healthy condition. Stumps should be removed, carious teeth filled or extracted, and tartar scraped away. When mercury is being taken, the mouth must be kept scrupulously clean. The condition of the kidneys must be ascertained by a careful examination of the urine. During the treatment, the patient must be urged to take plenty of good nourishing food, but alcohol must only be taken in strict moderation; hospital patients should be forbidden it. Chills must be guarded against, and flannel

should be worn next the skin. Regular action of the bowels must be ensured; and if anæmia is present, as is frequently the case, iron should be given as well as mercury.

It is extremely important that the patient be rapidly brought under the influence of mercury. For this purpose the drug may be administered by inunction, by inhalation, or by hypodermic injection, either intramuscular, intravenous, subcutaneous, or subconjunctival.

Method of inunction.—This, perhaps the best of all methods, is performed by rubbing into the skin, on the inner side of the arm or leg, 30 to 60 grains of the Unguentum Hydrargyri of the B.P. The skin should first be carefully washed with soap and water. The ointment must be rubbed in for about ten minutes every night just before retiring to bed, where the patient should be well covered with blankets to produce sweating. Each morning a warm bath must be taken. This treatment should be continued for about six weeks, and repeated, after an interval of about three weeks, for the same period. During the interval, and subsequently, mercury may be taken by the mouth in the form of the pill, or, better, in a mixture containing the perchloride combined with iodide of potassium.

Method of inhalation.—On alternate nights the patient is subjected to the vapour of calomel mixed with steam. Sitting on a cane-seated chair, and covered with blankets, he is surrounded by vaporised calomel, and caused to sweat profusely by the accompanying steam. Twenty to sixty grains of calomel should be used each time, and the baths continued until the patient is just short of salivation. They should then be stopped, and mercury continued to be given by the mouth, with or without iodide of potassium.

Method of injection.—The bichloride, biniodide, or cyanide is used for this purpose, not more than a quarter of a grain being injected at each time. Darier has extensively used subconjunctival injections of cyanide of mercury (1 in 2,000 to 1 in 5,000) for all deep-seated inflammations of the eyes, such as iritis, cyclitis, and choroiditis. Much pain follows in many cases; this, however, is greatly diminished if a few drops of a 1 per cent. saline solution of acoine be injected with the

mercury salt (see p. 135). Their action depends probably on the acceleration to the lymph current, which they produce, as well as on the antiseptic and antisiphilitic properties of the injections.

In whatever form the mercury is administered, it must be stopped at once if the gums become spongy, the mouth sore, or salivation excessive.

In the *rheumatic form*, salicin, salicylate of soda, or aspirin should be given. Iodide of potassium is sometimes very useful. Turkish baths are of great service. The pain in *gouty iritis* is sometimes completely relieved by colchicum; at other times the drug appears quite inert. The constitution should be treated upon proper dietetic principles, and alkalies freely used.

Tuberculous irido-cyclitis is rarely amenable to any local or constitutional treatment; and as it forms a centre for the dissemination of tubercular matter, it should be treated by prompt excision. If tubercular changes exist elsewhere, an attempt might be made to save the eye, though the precipitation of acute glaucoma will necessitate its removal.

Gonorrhæal iritis should be treated with potassium iodide by the mouth, together with local application to the urethra should any gleet be present.

4. *The diminution of any increase in tension* is very necessary in irido-cyclitis. The atropine must be discontinued, and either replaced by the use of eserine drops ($\frac{1}{2}$ per cent.), by paracentesis, or by iridectomy. The former of the two operations is often performed in suppurative iritis to relieve tension and to evacuate pus from the anterior chamber. The only other form of iritis likely to be attended with glaucomatous symptoms is tubercular iritis, the treatment of which has already been discussed.

5. *The treatment of complications and sequelæ.*—For *chronic iritis* iridectomy is the most reliable remedy; but, before resorting to operative measures, a sufficient length of time should be expended in the trial of milder means, such as change of air, particular attention to regular habits, an improvement of the general health by tonics, and by changing the mydriatic or stopping its use altogether. Iridectomy

should be, if possible, done upwards; it is usually followed by subsidence of all inflammatory signs and relief from pain.

When *posterior synechiæ* have formed, the number and extent of the adhesions will be shown by the effect of a $\frac{1}{2}$ per cent. solution of atropine dropped into the palpebral aperture. If only one or two points of attachment exist, they may be left alone and disregarded so long as the eye remains quiet; but should they be found to cause recurrent inflammatory attacks, the operation of iridectomy must be resorted to as the most reliable means of affording relief.

When *extensive synechiæ* or *total posterior synechia*, with or without *occlusion of the pupil*, are found to exist during or after an attack of iritis, their presence must be regarded as antagonistic to the welfare of the eye. By the dragging and limited movement thus imposed upon the iris, and by the obstruction constantly offered to the circulation of the intra-ocular fluids through their ordinary channel, the pupil, they are sure at some time to set up further trouble. This may appear in the form of recurrent inflammation, which often extends from the iris to the ciliary body and the choroid, or it may manifest itself by increased tension of the globe, either with or without inflammation—*secondary glaucoma*. Every possible effort must therefore be made to remove these adhesions, and to establish the circulation of the aqueous humour through the pupil. In the first place, by the use of strong mydriatics, such as atropine, and by the treatment of constitutional symptoms, much breaking down and re-absorption of the plastic exudation may sometimes be accomplished. If these means fail to liberate the iris from its adhesive bonds, the next step is to perform a free *iridectomy* without further delay. This should be done whether chronic recurrent iritis be present or not. The position of the section of the iris must depend upon the condition of the pupil; if this be occluded, the iridectomy should be made downwards and inwards, so as to give an artificial pupil; if the pupil be tolerably clear, the section may be made upwards. Von Graefe found this operation to be of the greatest service, not only in the reduction of inflammation and intra-ocular tension, but in the improvement of vision, and in the prevention of recurrent attacks. His

experience has been fully confirmed by others, and his practice is now generally adopted, with the best results.

THE OPHTHALMOSCOPIC APPEARANCE OF THE HEALTHY CHOROID.

The appearance of the healthy choroid must be carefully studied before we can properly appreciate the localised inflammatory and other changes which occur in the course of the diseases of that part of the eye.

We have seen (p. 174) that the choroid consists from within outwards of five layers of structure, which, for convenience of description, are called (1) the lamina vitrea, (2) the chorio-capillaris, (3) the lamina vasculosa, (4) the lamina suprachoroidea, and (5) the lamina fusca.

Now the colour of the *fundus oculi*, which is seen by reflected light when we use the ophthalmoscope, is due to three chief causes—viz. (1) the blood contained in the chorio-capillaris and lamina vasculosa; (2) the pigment-granules contained in the cells of the pigmented layer of the retina, and in the interstices of the vascular layer, besides the lamina fusca and lamina suprachoroidea; and (3) the sclerotic, which reflects a certain amount of light through the choroid and retina.

When this pigment is altogether absent from both retina and choroid, as is the case in *albinos*, we get a light yellowish-red colour, reflected from the blood within the capillaries; whilst the interstices between the latter are seen to be of a lighter, almost white, appearance, owing to the reflection from the sclerotic beyond the lamina fusca, and thus a fairly well-defined outline of the choroidal vessels is obtained.

In *fair persons*, where the pigment-granules contained within the cells are only of a faintly brown colour, the fundus has a yellowish-red colour, and the vessels of the choroid can often be seen, although less distinctly than in *albinos*.

In *moderately dark persons* this pigment becomes of a deeper brown, and the fundus presents a light brownish-red colour, no choroidal vessels being seen (see figs. 1 and 2, on the opposite page). If, however, as is occasionally the case, pigment is scarce in the pigmented layer of the retina, while abundant in the choroid, a network of red vessels is seen upon a dark background (*choroïde tigrée*).

In *negroes*, and all *dark races*, the pigment is so abundant as to prevent the appearance of almost all red reflex from the choroid, the fundus assuming a brownish-grey, or even slate colour.

The colour of the fundus varies very much with the intensity and colour of the light used, with the state of dilatation of the pupil, and

Plate II.

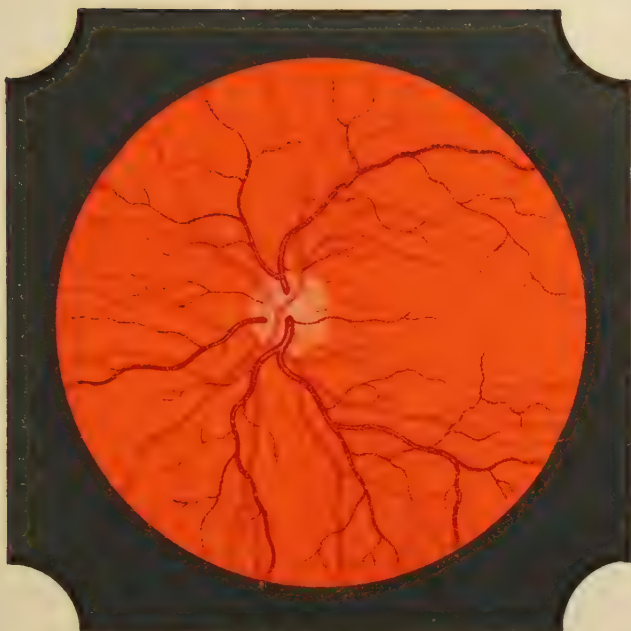


Fig. 1.—Normal fundus.

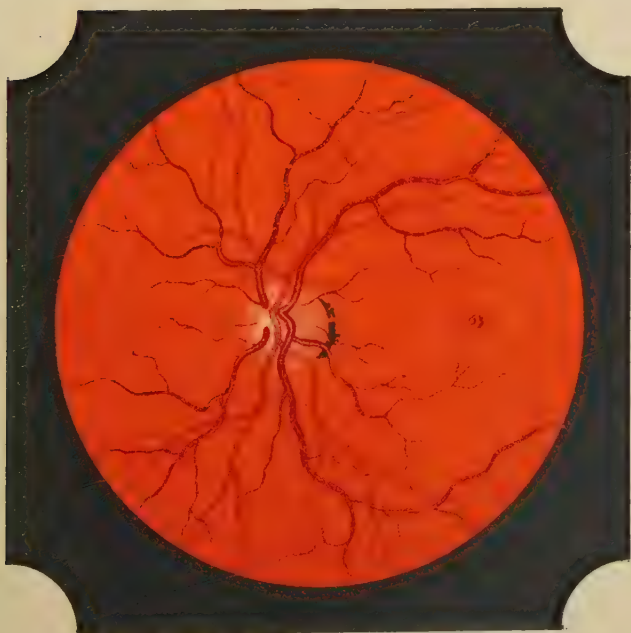


Fig. 2.—Normal fundus.



with the refractive condition of the eye. It is brighter, *cæteris paribus*, in proportion to the number of rays of light that can be thrown into the eye.

HYPERÆMIA OF THE CHOROID.

This condition has for its chief ophthalmoscopic sign a blushed appearance of the disc, with diminution in its differentiation from the rest of the fundus. It may be the first stage of a threatened papillitis, but more commonly it denotes eye-strain, usually due to uncorrected hypermetropia. It should be treated by rest, produced by atropine and dark glasses, with subsequent correction of any existing error of refraction.

CHOROIDITIS.

Inflammation of the choroid is accompanied by infiltration and exudation, which may be re-absorbed with or without atrophic changes, or may pass into the stage of suppuration. If the morbid process is limited to the choroid proper it is frequently unattended by external signs of inflammation, being recognised chiefly by visual troubles and ophthalmoscopic appearances. When the other portions of the uveal tract—the ciliary body and iris—are involved in the process, as is usually the case in acute and suppurative forms, the external signs of these inflammations are always present. The two chief varieties of choroiditis are *plastic* or *exudative choroiditis* and *suppurative choroiditis*. Besides these, there are the forms accompanied by inflammation of neighbouring parts; these are *irido-choroiditis*, *retino-choroiditis*, and *sclerotico-choroiditis*.

I. Plastic or Exudative Choroiditis.—*Symptoms.*—Plastic choroiditis is the most common variety, and presents itself in several forms, the symptoms varying with the extent of the area affected and with its position with regard to the macular region. When unattended with cyclitis or iritis, there are no external signs of the disease, the circumcorneal zone is not injected, the cornea is clear and free from keratitis punctata, the pupil is normal in shape and colour and responds to light. *Subjective symptoms* are usually the earlier and more important indications of the disease, but they may be completely absent although the ophthalmoscope shows

gross changes. The patient complains of seeing lights of a pale-blue or red colour—*phosphenes*—at night, when in bed and the eyes are closed and the room dark. In the daylight he sees large floating specks, especially when looking at a white object such as a sheet of paper; these are larger than the ordinary *muscæ volitantes*, and tend to obscure the vision by settling upon one portion of the paper looked at. Distortion of outline of objects—*metamorphopsia*—is also a characteristic feature, especially when the exudative change is situated in the macular region; if parallel straight lines are held in front of the eye, they will appear to be curved in various ways. *Micropsia*—objects appearing smaller than usual—and *macropsia*—objects appearing too large—are symptoms sometimes present. The patient may complain of dark spots in the visual field—*positive scotomata*—or these may only be found by carefully testing the field with the perimeter—*negative scotomata*. It will then be found that its whole area is not intact, but that, according to the position and extent of the disease, there will be small and large areas in which vision is either defective or altogether absent—*relative* or *absolute scotomata*. The light-sense is also appreciably diminished in choroiditis, especially when there is cloudiness of the vitreous. In many cases the patient will complain of a dull aching pain at the back of the eyes, but this symptom is never pronounced and may not be noticed.

Ophthalmoscopic signs.—Recent patches of choroiditis appear as yellowish ill-defined areas lying beneath the retinal vessels upon and in the red background of the choroid. These patches may be more or less obscured by a hæmorrhage from a choroidal vessel, this being sometimes the earliest sign of the disease. This yellow exudation may entirely disappear after some weeks. In rare cases, and under proper treatment, it leaves the choroid intact; but more commonly, as it disappears, the affected area is found to be more or less atrophic, gradually becoming whiter, with the choroidal vessels showing up, pigment at the same time appearing round each patch and within some of them; crystals of cholesterin may be seen on some of the patches. Still later, the choroidal atrophy presents the appearance of white patches, ringed by pigment,

Plate III.

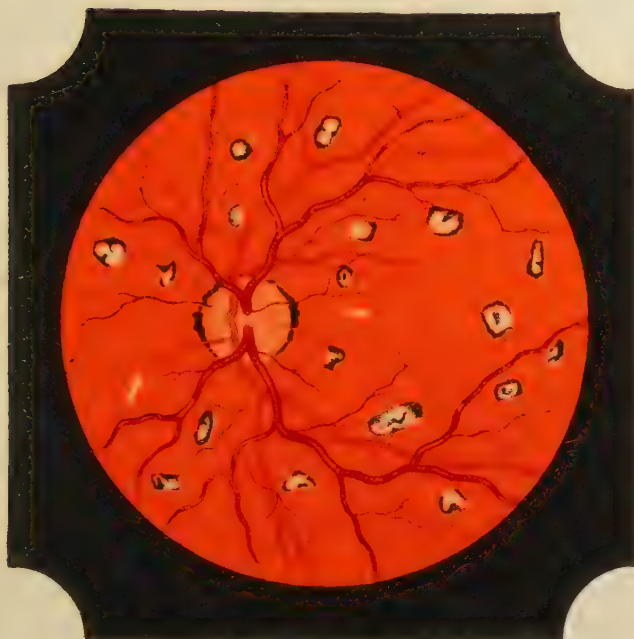


Fig. 1.—Disseminated Choroiditis.

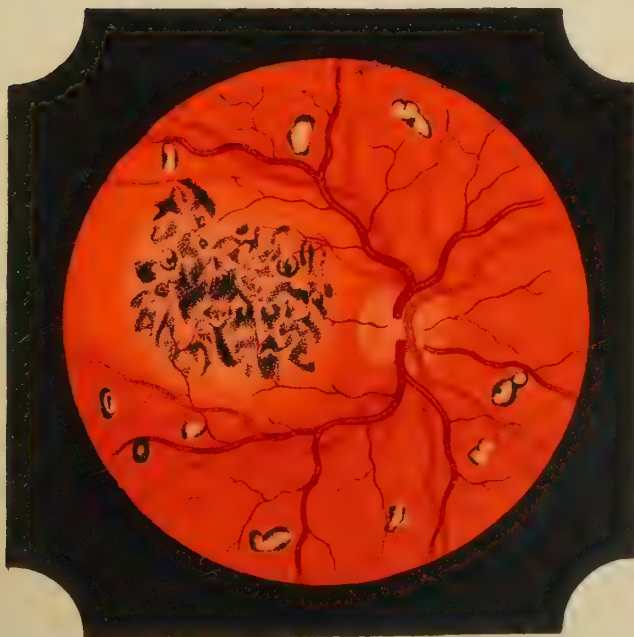
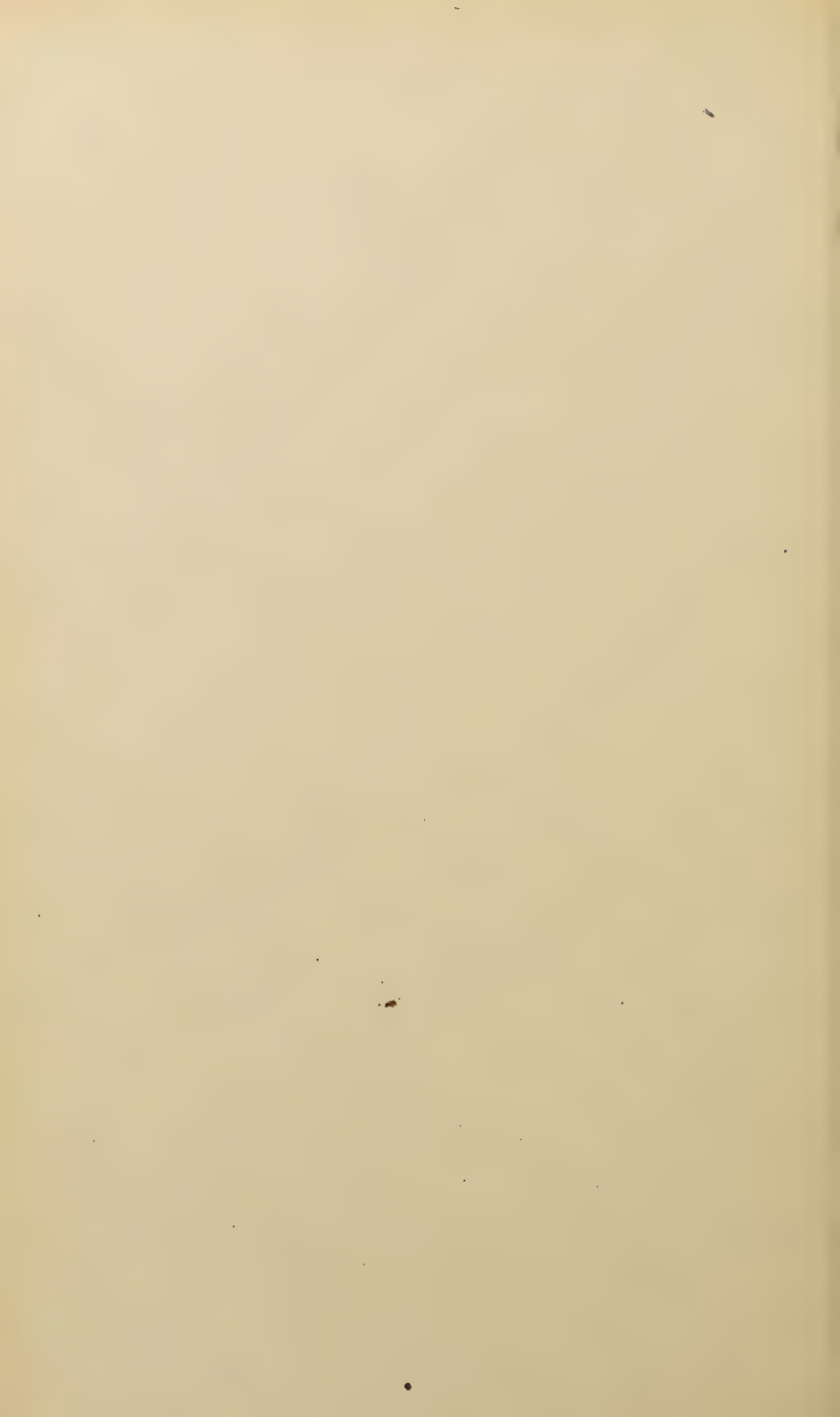


Fig. 2.—Disseminated and Central Choroiditis.



with no trace of choroidal vessels, but crossed uninterruptedly by the retinal vessels. Fig. 1, opposite p. 200, represents the characteristic features of a fairly advanced stage of plastic choroiditis; whilst fig. 2 shows a more advanced case of the same disease. It is not uncommon to find both old and recent patches in the same eye. The inflammation does not limit itself to the choroid, but usually attacks the adjacent retina; indeed, the exudation often extends quite through the retina and into the vitreous, in front of each patch. The vitreous is therefore often in a cloudy condition owing to the presence of fine opacities. This is more particularly the case in syphilitic choroido-retinitis. The nebulous condition of the vitreous not only interferes with the patient's vision, but prevents the details of the fundus from being clearly made out with the ophthalmoscope; the existence of cloudiness of the vitreous is therefore always suggestive of choroiditis being present, although hyalitis without choroiditis is sometimes found.

Etiology.—The most common cause is syphilis, inherited or acquired. In the acquired form it usually occurs towards the end of the first year after the primary infection, or during the beginning of the second year. Inherited syphilitic choroiditis is commonest between the ages of six months and three years; it may occur, however, much later, together with interstitial keratitis. Other causes of plastic choroiditis are tuberculosis, gonorrhœa, the simple and profound anæmias, and menstrual disorders. Many chronic cases occur to which no definite cause can be assigned.

Pathology.—When a recent patch is examined microscopically, we find a cluster of round and fusiform cells in the region of the lamina vitrea and the chorio-capillaris, with great engorgement of the vessels, and perhaps one or two hæmorrhages. The pigment-layer is then unaffected. As the disease progresses, the cells of the pigment-layer begin to proliferate, and the part which is immediately opposite to the patch becomes absorbed, giving it a white appearance; the pigment becomes accumulated at the edges of the patch, and the inflammation extends to the layer of rods and cones, and the outer granular layer of the retina. Later on, this inflammatory exudation becomes absorbed, and gives place to

cicatricial tissue; but the structures involved—viz. the outer granular layer, the rods and cones, the uveal tract, and the vessels of the choroid—are found to be destroyed, and their place occupied by this new cicatricial connective tissue.

Varieties.—Many different kinds of exudative choroiditis are found. The following may be mentioned as those more frequently met with: (1) Disseminated; (2) Central; (3) Peripheral; (4) Diffuse exudative; (5) Myopic.

(1) *Disseminated choroiditis* consists of numerous round or irregular spots scattered over the fundus (fig. 1, opposite p. 200). These exudations may be of the same or of different ages. They pass through the changes mentioned above, and mostly end in patches of complete atrophy. They may finally become confluent, involving the whole or greater part of the fundus. The optic nerve may be involved in the process, in which case it becomes first hyperæmic and finally atrophic. This form of choroiditis may easily be mistaken for a peculiar condition of the choroid, first described by Waren Tay as *guttate choroiditis* (p. 237).

(2) *Central choroiditis* consists of exudation in the macular region, forming a small or large irregular, more or less circular, patch, with considerable pigmentation. Vision is much deteriorated, and a large central scotoma is present. This is especially frequent in old people, where a very chronic inflammation, without much pigmentation or exudation, is followed by well-defined atrophic changes. In such cases the fenestrated arrangement of the choroidal vessels is clearly seen. This senile form is usually bilateral, and must be carefully sought for before the extraction of all senile cataracts, as its presence will greatly modify the prognosis.

(3) *Peripheral or anterior choroiditis* is that form in which the central portions of the choroid are unaffected, whilst the peripheral parts are interspersed with the diseased patches. Owing to the outlying position of the lesion, the direct and ordinary vision is but little disturbed, and the disease may on this account be easily overlooked unless the peripheral portions of the fundus are carefully examined with the ophthalmoscope.

(4) *Diffuse exudative choroiditis*, extending over the whole fundus. Although the whole choroid is affected, yet the deposit

Plate IV.

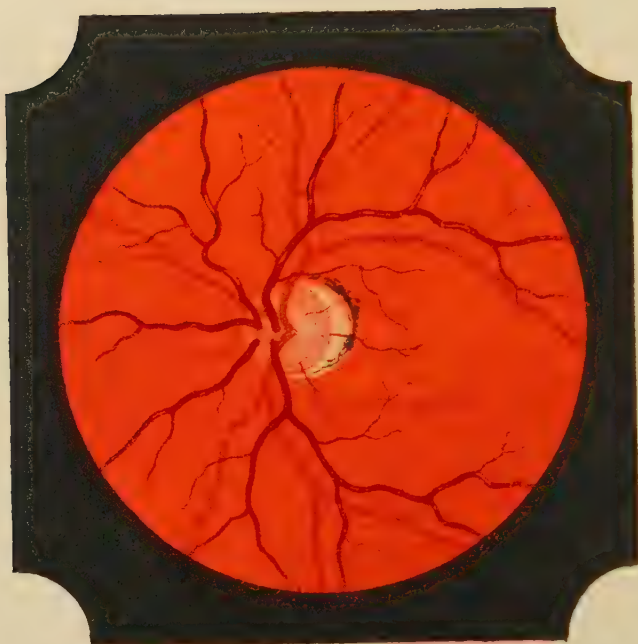


Fig. 1.—Myopic Crescent.

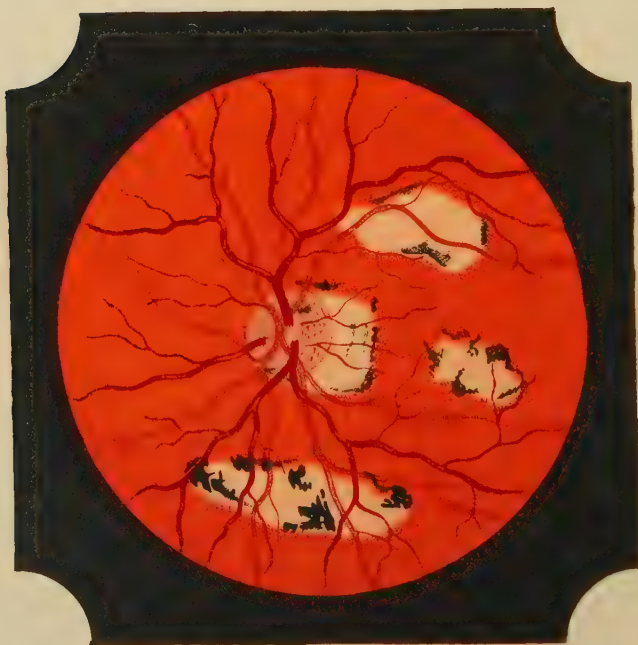


Fig. 2.—Posterior Staphyloma.
Patches of Atrophy following Choroiditis.

of lymph appears to assume the form of circumscribed patches, varying somewhat in tint from a yellowish-red to white, with pigmentation according to the stage of the affection. It is always more or less obscured from view by the presence of fine 'dust-like' opacity of the vitreous, and not unfrequently large membranous floating opacities of the vitreous are present. The vitreous and the retina may ultimately become clear, and then the ophthalmoscope reveals more definitely the large atrophic patches in which masses of pigment are here and there distributed. Such cases, when they have arrived at an advanced stage, are often difficult to distinguish from *retinitis pigmentosa* (see p. 293), for the retinitis secondary to syphilitic choroiditis tends to simulate that disease. Peripheral retinal pigmentation, night-blindness, and contraction of the visual field occur; and it is only by the presence of *vitreous opacities*, patches of *choroidal atrophy*, and *relative distension of the veins*, together with perhaps concurrent iritis or cyclitis, and a definite history of syphilis, that the diagnosis can be arrived at. Retinitis pigmentosa, again, is always bilateral, whereas diffuse exudative choroiditis may be unilateral. The visual field, too, may help, for in some instances there is no peripheral contraction, but a *ring scotoma*, which is almost pathognomonic of syphilitic choroido-retinitis.

This kind of choroiditis usually affects the retina to a serious extent; the rods and cones are quite destroyed, and the pigment arranged in heaps, whilst the choroidal tissue shows signs of infiltration with lymph-cells and cicatricial contraction.

(5) *Myopic choroiditis*. See Sclerotico-choroiditis, p. 209.

Diagnosis.—The exudation into the vitreous often completely obscures the fundus from view. In these cases the nature of the vitreous opacities and the history of the case are all the signs. As the opacities disappear a patch of choroidal atrophy will be discovered. Care must be taken to exclude a nebulous cornea, as this will cause a haziness of the fundus very similar to that produced by fine vitreous opacities. On the other hand, a hazy vitreous will make the outline of the optic disc appear blurred, and thus simulate papillitis.

Prognosis.—Plastic choroiditis runs a chronic course; its

worst feature is a tendency to recurrence. Many cases are so chronic that they extend over years without perceptible change; whilst others present new spots of exudation from time to time, until the choroid is ultimately covered with atrophic patches. In bad cases the optic nerve and retina are involved, and partial or complete blindness results. Vision, however, may be almost completely restored as long as the macular region is free. Central changes, like peripheral, are attended with subsequent atrophy, which gives rise to permanent and absolute scotomata; consequently, direct vision becomes completely lost. Diffuse exudative choroiditis is invariably attended with gradual failure of sight, until complete blindness ensues.

Treatment.—It is necessary to find out, if possible, the cause of the disease. A history of syphilis can in many instances be elicited; and should the disease be active, mercury in some form must be administered either with or without iodide of potassium (see p. 194). Other constitutional causes require general treatment. Locally, if the condition is in an active stage, as shown by dull pain, diffuse vitreous haze, and fresh exudations, leeches applied to the temple, diaphoresis by means of hot-air baths or pilocarpine hypodermically injected, and profuse purges are necessary. Counter-irritation by means of the Argyll-Robertson method frequently helps to clear up the condition. This consists in rubbing into the skin of both eyelids pure silver nitrate until vesication is produced. The eyes should be protected from the light by the use of tinted glasses; they should be rested as much as possible, and no near work be attempted. It may be necessary to atropinise the eyes to prevent accommodation. Should there be no history of syphilis, it is, in many cases, advisable to try the mercurials and iodides, as great improvement is often obtained by their use independent of any syphilitic history. Sub-conjunctival injections (see p. 195) in early cases are often useful. Even after the disease has apparently reached the complete atrophic stage, potassium iodide should be persevered with, as marked improvement in visual acuity often takes place in what seem to be hopeless cases. The general health must be supported by a nutritious diet, regular outdoor

exercise, and tonics, as iron, strychnine, quinine, &c.; local treatment must also be adopted as indicated above. It is advisable in most cases of choroiditis to avoid alcoholic stimulants.

II. **Suppurative choroiditis** is a diffusé suppurative inflammation of the choroid, and is always attended with suppurative irido-cyclitis. There is, in fact, a general inflammation of the tunica vasculosa, which usually spreads to the other tunics of the globe and gives rise to that condition known as *panophthalmitis*.

The acute and chronic forms of this disease must be considered separately.

Acute Suppurative Choroiditis.—*Etiology.*—The causes of acute suppurative choroiditis may be divided into three groups: (a) Injury of a penetrating nature, if septic infection by pyogenic organisms is produced. Operative measures on the eyeball, where rigid aseptic precautions have not been taken, may be placed under this category.

(b) There may be an extension of inflammation elsewhere, such as from a septic ulcer of the cornea.

(c) Inflammation of the choroid may be the result of metastasis. Of metastatic choroiditis, the most important is the puerperal form, occurring as a symptom of pyæmia, usually in the second week after delivery. It is also occasionally found in ulcerative endocarditis, cerebro-spinal meningitis, and during the acute specific fevers, especially typhoid fever, pneumonia, and influenza. It is due to septic embolism of the retinal and choroidal vessels.

In some cases of suppurative choroiditis it is difficult to trace the cause.

The symptoms from the first are those of intense inflammation. The conjunctiva and subconjunctival tissues are densely infiltrated with serum, so that the cornea is partly covered in by the swollen tissues. The eyelids, also, are red and swollen; so much so, that were it not for the absence of discharge the case might be considered to be one of purulent conjunctivitis. The iris is changed in colour, and becomes muddy in appearance, the pupil fixed, and the cornea hazy and anæsthetic. The globe of the eye appears swollen and pushed forwards; it is hard to the touch, and extremely painful on pressure. There

is excessive pain, at first in the eye, and afterwards in and around the orbit. Vision is of course soon diminished, and finally lost altogether. Pus forms in the anterior chamber, and is accompanied by general pyrexia. If the media are sufficiently clear, the exudation into the vitreous can be seen as a yellowish reflex.

As time goes on, there may be a gradual subsidence of the symptoms, the proptosis diminishes, the inflammation subsides, the tension falls, and gradually a general shrinking of the whole globe (*phthisis bulbi*) occurs. In the acute form, however, suppuration is accompanied by marked constitutional symptoms. High fever, vomiting, and violent pains ensue; the proptosis increases, the lids become more swollen, until the eyeball gives way anteriorly, with escape of pus and disappearance of the acute symptoms.

Pathology.—Suppurative irido-choroiditis is attended with suppurative retinitis; the vitreous, also participating in this progressive and destructive inflammation (suppurative hyalitis), becomes destroyed and replaced by pus, and the eyeball is converted into an abscess-cavity the tunics of which form its wall. In such a state one of two terminations will occur—either the cornea will slough and the pus be discharged, or the pus will become inspissated by absorption of its liquor puris; in either case, the globe will shrink. In many cases the exudation in the vitreous is more puriform than purulent (see p. 207).

Microscopically, the choroid will be found to be several times its natural thickness and exceedingly cellular, the cells aggregating into clusters which form small abscesses; these soon coalesce and convert the choroid into a diffuse suppurative tract. The inflammatory changes in the iris, ciliary body, retina, and vitreous are similar to the microscopical characters of inflammation in other parts of the body. It is advisable to mention, however, that the partially organised exudation in the ciliary body looks under the microscope not unlike a melanotic sarcoma, on account of the complete derangement and multiplication of the pigment-cells. The clinical history, together with the condition of the vessel-walls, will explain the nature of the growth.

In metastatic choroiditis it is often possible to find the infecting emboli, which can be shown to contain pyogenic organisms, the streptococci being the most common.

Treatment.—Local leeching, hot fomentations and poultices, combined with morphia, either hypodermically or otherwise administered, are very useful in allaying pain. As soon as pus is evidently accumulating in or behind the aqueous chamber, prompt surgical interference is indicated. If the eye be left to itself, there is considerable risk of the inflammation extending backwards along the optic nerve to the brain and its membranes, and so causing a fatal termination. Excision of the globe is, in my opinion, the best and safest way of treating this severe condition. Some surgeons, however, are doubtful as to the propriety of removing an eye whilst in this inflamed and suppurating condition, and prefer first to make an incision through the anterior part of the globe so as to relieve pain, tension, &c., and to postpone the excision until the inflammatory symptoms have subsided.

Subacute and Chronic Suppurative Choroiditis.—In some cases exudation into the vitreous is puriform rather than purulent, the choroiditis being of a subacute or chronic nature. This condition is usually found in children, and, since it often closely resembles in appearance glioma retinae (see p. 307), is of extreme importance.¹

Etiology and pathology.—Middle ear disease is a very usual accompanying symptom, and this, together with the frequently found history of fits or convulsions at some previous time, with perhaps retraction of the head, point to posterior basal meningitis as being in some way connected with this disease. It is uncertain what is the exact relationship between the two conditions. The ophthalmitis may be a direct result of the meningitis, or they may both be the result of a pyæmic infection, starting from otitis media. Other etiological factors seem to be the acute specific fevers, and possibly congenital syphilis.

The exact seat of the primary changes in the eyeball appears to be the choroid, though the retina and optic nerve

¹ For this reason the term *pseudo-glioma* has been largely used to designate this disease; see footnote on p. 307.

are soon affected. There may be a diffuse inflammation of the whole uveal tract. With subsidence of active inflammatory changes, the puriform exudation only very slowly undergoes organisation, and consequently the ophthalmoscopic condition may remain for many weeks unchanged. Eventually, however, the eyeball enters into a state of phthisis bulbi, and in some cases the choroid undergoes partial ossification.

Clinical features.—By oblique focal illumination, and also upon ophthalmoscopic examination, a dull greyish-white flocculent-looking reflex is seen in the pupil. The red fundus reflex is usually absent. There may be signs of active or recent iritis, though this is by no means always the case. The tension of the globe may be normal, but is usually diminished.

Diagnosis.—The diagnosis from glioma retinae is considered on p. 307.

Prognosis.—A fatal termination is unusual; this is probably because the meningitis is not, as a rule, extensive. The eyeball eventually shrinks and becomes puckered and hardened.

Treatment.—As far as the eye is concerned, nothing can be done. In all cases, however, even though there appears to be no otitis media, both tympanic membranes should be examined with a view to puncture.

III. *Irido-choroiditis* (uveitis) is an inflammatory condition involving the whole uveal tract, the iris, ciliary body, and choroid. It may be *acute* or *chronic*.

Acute irido-choroiditis is either suppurative (see Suppurative Choroiditis) or sero-plastic (see Sympathetic Ophthalmitis).

Chronic irido-choroiditis is a slow form of inflammation which attacks each part of the uveal tract either simultaneously or successively. It is very often preceded by an acute attack of iritis or choroiditis. Subacute exacerbations occur from time to time. Its tendency to relapse is its predominating feature; years may pass away before there is a permanent cessation of inflammatory symptoms. The prevailing clinical features are evidences of iritis, cyclitis, and choroiditis, such as posterior synechiæ or pigment on the lens capsule; thinning of the iridic tissue so that in some cases the dull choroidal reflex may be seen through the reticulum of the iris as if through a curtain;

thinning of the sclera over the ciliary body, from chronic cyclitis, which allows the blue ciliary body to be seen through; eventually a ciliary staphyloma may appear. Numerous and large vitreous opacities are seen; and, if the vitreous is not so cloudy as to obstruct the view of the fundus, patches of choroiditis and choroidal atrophy, with general choroidal thinning, may be made out. Posterior polar opacity or irregular opacities in the lens are not uncommon, and at last the whole lens may become cataractous. Subjective signs are present in the form of defective vision, sometimes so bad as to be only perception of light, flashes of light in the dark, dull aching pain in the eyes, &c. The tension is usually slightly raised at first, but soon becomes subnormal ($T-1$ or $T-2$). The prognosis is unfavourable. The disease is usually symmetrical, and complete blindness will almost certainly ensue sooner or later.

Treatment.—Little can be done beyond treating the symptoms as they appear, and supporting the general health. Iodide of potassium assists in some cases, chiefly on account of its alterative action and its power in aiding the absorption of all chronic inflammatory exudations. Mercurial inunctions are also useful for the same purpose. Iritis must be treated in the early stage by instilling atropine and cocaine. Increase of tension is rarely present, and is certainly never sufficiently pronounced to necessitate operative interference.

IV. **Retino-choroiditis.**—There is almost always some accompanying retinitis with all cases of plastic choroiditis. It is unnecessary to add anything to what has been said under that heading.

V. **Sclerotico-choroiditis or Myopic Choroiditis.**—This is rather of the nature of an atrophic condition of the choroid than a true inflammation, and will be described under Myopia (see p. 491).

TUBERCLE OF THE CHOROID.

Tuberculous disease may affect the choroid in three ways, which differ in ophthalmoscopic signs, prognosis, and treatment.

(a) **Tuberculous Disseminated Choroiditis.**—This form of chronic inflammation of the choroid is very similar to the syphilitic variety, and is, according to some authorities, as

common. It is with difficulty distinguished, but the patches are smaller and slightly raised. Just as in the syphilitic form, degeneration takes place with choroidal atrophy, the process being, however, slower. Its treatment is unsatisfactory; before atrophic changes have set in, mercurial inunction may help absorption of the exudation, and phosphates or arseniates with cod-liver oil should be prescribed by the mouth. The prognosis is not so good as in the syphilitic variety.

(b) **Miliary tuberculosis of the choroid** occupies the region of the chorio-capillaris and the vascular layer, and is quite behind the uvea. It is most commonly found in cases of acute miliary tuberculosis, but it may be present in all forms and stages of tuberculous disease.

Ophthalmoscopically it appears as a greyish hemispherical eminence, varying from 3 mm. to 1 mm. in diameter, and may be even smaller. One or several of these first appear in the macular region, and they are followed by others in the immediate neighbourhood. The youngest tubercles are very small; the oldest are the largest, and somewhat white at the centre. The ophthalmoscopic signs are very similar to those of disseminated choroiditis, though the two conditions are not likely to be mistaken, owing to the different general conditions. The patches are more raised, rarely pigmented, more clearly defined, and less brilliantly white. In the form that occurs in acute general tuberculosis, they appear, as a rule, only a short time before death. Papillitis is a frequently accompanying symptom. Both eyes are nearly always affected.

Microscopically, each patch shows the typical structure of tubercle, though the specific bacilli are not always to be found.

In cases of acute tuberculous disease where there are typhoid symptoms, and in tuberculous meningitis where the diagnosis is not always easy, the detection of tubercle of the choroid is of great assistance in clearing up the case, although the absence of choroidal tubercle does not prove the absence of tuberculous disease in other organs.

(c) **Tuberculous Tumour of the Choroid.**—This condition is probably always secondary to tuberculous disease elsewhere, though frequently this cannot be proved. In early stages, a retinal detachment, less defined than the detachment due to choroidal

sarcoma, will be the only sign. As growth takes place glaucomatous symptoms rarely occur, the coats of the globe rapidly giving way with the formation of a staphyloma and subsequent panophthalmitis. It is doubtful if general infection can occur, so enucleation should be postponed until vision is lost. The growth is always unilateral.

GUMMA OF THE CHOROID.

Gumma of the choroid is very rare. It may exist in conjunction with gumma of the iris or ciliary body. The diagnosis is necessarily difficult, inasmuch as the vitreous is hazy and the fundus cannot be seen. It rests mainly on the presence of concurrent syphilitic lesions, on the history and on the effect of antisyphilitic treatment. Sight is usually greatly impaired, but, if the condition is attacked in time, almost perfect vision may be regained.

SYMPATHETIC IRRITATION AND SYMPATHETIC OPHTHALMITIS.

These terms are applied to certain affections which are set up in one eye in consequence of some organic lesions of its fellow on the opposite side. The eye which is first affected is usually spoken of as the *exciting eye*, whilst the second is called the *sympathising eye*.

Etiology and Pathology.—In the exciting eye there is almost always a history of an injury at some time or other. In the majority of cases this has been a penetrating, incised, lacerated, or contused wound of the *ciliary region*. It occasionally happens that the wound produced by a blow is subconjunctival, and so may escape the notice of the surgeon. Penetrating wounds of the eyeball which injure the ciliary body and also the lens, thus causing a swelling of this latter structure, are very prone to set up sympathetic ophthalmitis.

The presence of a foreign body lodged within any part of the globe, such as a shot or a chip of metal, is very likely to cause disorganisation of the injured eye, and is a very probable forerunner of sympathetic trouble in the other.

Wounds of the cornea which do not extend to the ciliary

region have of themselves little tendency to set up sympathetic inflammation; but should they be attended by dislocation of the crystalline lens, or by the formation of anterior synechia, these lesions are very liable to produce it.

Besides injuries, another condition of the exciting eye has been known to cause sympathetic ophthalmitis. Intra-ocular growths may set up an irido-cyclitis in the second eye, but probably only if irido-cyclitis is present in the first eye.

In the case of spontaneous inflammation of one eye, followed by similar symptoms in the other—as, for example, chronic irido-choroiditis, ciliary staphyloma, glaucoma, &c.—it is difficult or impossible to prove that the affection of the second eye is due to an extension of the disease from the first, and not to a common cause. An eye, however, which is shrunk and disorganised is very liable to take on an inflammatory condition which may cause irritative symptoms in its fellow, and such an eye should therefore always be regarded with suspicion, especially when the other eye is in any way irritable, or inflamed without apparent cause.

The *mode of production* of sympathetic disease has yet to be explained. The oldest theory, which goes by the name of Mackenzie's, is that the inflammation spreads to the sympathising eye along the optic nerve and chiasma. More recently it has been held (H. Müller) that the ciliary nerves formed the conducting paths, the inflammation being conveyed to a centre of the ciliary nerves of the injured eye, and thence reflected down the ciliary nerves of the other eye, or else that the vaso-motor centre was acted upon in such a way as to interfere with the nutrition of the other eye. Pathological proof in support of this theory is wanting; the material, however, is not plentiful, and it would be easy to overlook morbid changes in nerves so minute and numerous as are the ciliary nerves. A more powerful argument against the reflex production of sympathetic ophthalmitis lies in the fact that true inflammation has never been produced experimentally by irritation of a nerve.

It appears probable that sympathetic ophthalmitis has an infective origin. There are, however, many difficulties to this theory. In the first place, the specific organism has not been discovered. The ordinary pus micro-organisms rarely cause

the disease; only a few cases of sympathetic ophthalmitis following panophthalmitis have been recorded. The observations of some experimenters have shown that the space between the dural and pial sheaths of the optic nerve, and the lymph sheaths surrounding the arteries of the retina and of the optic nerve, are sometimes occupied by a number of lymphoid cells, similar in nature to those which are so abundant in the uveal tracts of the eyes affected. This has led to the theory that it is along these lymphatic spaces that the morbid process extends to the second eye; the chain of evidence is, however, incomplete, for it has not been shown that the chiasma is affected; while the fact that in some of the cases examined the changes have become less marked in each nerve as the chiasma was approached, renders it quite possible that the change observed indicated a morbid process extending backwards from *each* eye; lastly, many observers have completely failed to find any of these changes. The germs do not appear to enter the general circulation, as they otherwise would be liable to be introduced through a wound in other parts of the body; and of this there is no evidence, no case having been recorded. An autotoxic origin has been suggested, and this would explain those cases of sympathetic disease which have followed non-perforating injuries. The known facts, however, about the occurrence of sympathetic ophthalmitis are hardly yet sufficient to establish a theory as to its mode of transmission; before this can be done more data must be collected as to the essential nature of the injury which gives rise to it, the shortest interval that can elapse between the receipt of the injury and the appearance of symptoms in the other eye, and, above all, as to the exact nature of the morbid changes in all the possible paths in *both* eyes.

In the majority of cases where a wound of the ciliary region is followed by such a condition as to set up sympathetic inflammation, we find the presence of *plastic inflammation* of the iris, the ciliary body, and the choroid (irido-cyclo-choroiditis). During the first week after the infliction of the wound a violent reaction is set up, in which there is intense pain in the eye, orbit, and the surrounding temporal, frontal, and malar regions. There is marked congestion of the circumcorneal

zone of vessels, and the ciliary region is tender when digital pressure is made through the closed eyelids. There is great intolerance of light and overflowing of tears. The vision is much impaired. These symptoms are succeeded by those of chronic irido-cyclitis. The iris becomes extensively adherent to the capsule of the lens, it is changed in colour, and the pupil may be occluded with organised lymph. The vitreous, when the pupil is not occluded, is found to be so hazy, and crowded with opacities, as to prevent the retina and choroid from being seen with the ophthalmoscope. If the lens has been wounded in the accident, it of course becomes swollen and opaque. Upon section and microscopic examination of an eye in this condition of traumatic irido-cyclitis, from five to ten days after the infliction of the wound, we find evidence of severe plastic inflammation in the iris, ciliary body, and choroid. The *iris* is thickened by infiltration with lymphoid cells, which are arranged in clusters. These first appear in the middle strata, and then coalesce, and extend to all the tissue of the iris. The blood-vessels appear to be blocked by white corpuscles, and their walls are thick and translucent. The pigment-layer at the back of the iris is altered in appearance, its cells having undergone proliferation; it is less dark in colour, and there is a thick deposit of lymphoid cells on its posterior surface. The *ciliary body* is similarly affected. Clusters of cells first appear on the inner surface of the ciliary muscle; these increase and coalesce until they occupy the entire part between the muscle and the pigment-layer. The pigment-layer, again, is much altered, and appears merely as an irregularly scattered line in the midst of lymphoid cells. The fibres of the ciliary muscle are not much infiltrated. The *pars ciliaris retinæ* is but little altered, except that it is separated from the basement-membrane by exudation. The *choroid* also shows clusters of leucocytes, first appearing in the middle or vascular layer, which multiply, coalesce, and finally occupy its whole extent. Its thickness may be increased eight or ten times. The blood-vessels are blocked with leucocytes, and are ultimately destroyed. The pigment-layer is not affected. In the lymph spaces around the blood-vessels of the retina clusters of lymphoid cells are also sometimes seen. Similar

cells are also found in the intersheath space, and around the vessels of the optic nerve.

In some cases the active inflammatory changes in the exciting eye, as above described, are much less marked.

Although sympathetic ophthalmitis is usually an iridocyclitis, occasionally it takes the form of a retino-papillitis. This variety is much less malignant in character, and almost invariably slowly disappears, especially after removal of the exciting eye.

Symptoms.—It is important to distinguish between *irritation* and *inflammation* of the sympathising eye.

By *sympathetic irritation* is meant a functional derangement only. It is characterised by intolerance of light, lachrymation, and inability to use the eye for reading or work for more than a short period without a sense of fatigue, the power to accommodate being rapidly lost. The vision may be normal, but is often impaired. There are sometimes temporary sensations of darkness (obscurations) which last for several seconds. There may be considerable neuralgic pains in and around the eye. No physical signs of inflammation can be detected, either with the oblique focal illumination or with the ophthalmoscope. Sympathetic irritation frequently precedes inflammation, but it may exist for weeks, months, or even years before the inflammation of the eye supervenes. It is not a necessary forerunner of sympathetic inflammation, inasmuch as the latter often comes on without any symptom of irritation; on the other hand, it often subsides, and is not followed by sympathetic inflammation. The excision of the exciting eye usually causes speedy removal of the symptoms of irritation.

Sympathetic ophthalmitis may, as we have just seen, be ushered in by irritation, but it may come on in the most insidious manner, without pain, photophobia, or lachrymation; it usually commences as a *serous irido-cyclitis*, the inflammation thence extending to the choroid. The symptoms of this early stage are easily overlooked; but when carefully examined the pupil is found to be sluggish, the vision more or less impaired, the vitreous hazy so as to prevent a clear view of the retina and choroid. Floating opacities of the vitreous are often

present. Dots of opacity on the back of the cornea (keratitis punctata) can usually be seen as soon as this disease has fully set in. These are sometimes extremely minute, and then can only be seen by using a magnifying lens with the oblique focal illumination (p. 109). The dots are either scattered irregularly over the surface, or they assume a triangular arrangement, the apex of which is opposite to the pupil, and the base either below or on one side. The serous inflammation may continue as such throughout the whole course of the disease, or it may at any time assume the more severe plastic form. In the majority of cases of long-standing sympathetic disease, we find both the clinical and anatomical characters of *severe sero-plastic inflammation of the whole uveal tract*. The iris looks thick and fleshy, and is changed to a buff or brownish-yellow colour. Its blood-vessels become large and visible. The pupil may be blocked by lymph. Adhesions between the iris and anterior capsule of the lens are common. The vitreous, when visible, is found to be hazy, and to contain floating opacities. The zone of vessels around the cornea is intensely congested. There is sometimes intense neuralgic pain in the regions supplied by the fifth nerve.

The *interval of time* between the injury of the exciting eye and the onset of inflammation in the sympathising eye is very variable. It is seldom less than three weeks. The usual period is from eight to twelve weeks, but cases not unfrequently occur after a much longer period, even many years from the date of the original injury or disease; but there must of necessity be some considerable uncertainty as to whether the original injury was the real cause of these cases.

Diagnosis.—The diagnosis of sympathetic ophthalmitis is rarely difficult. A virulent attack of irido-cyclitis coming on with little or no pain three or more weeks after a perforating injury of the other eye, leaves little doubt as to the nature of the disease. Keratitis punctata is very frequently the first sign.

Prognosis.—The prognosis of sympathetic ophthalmitis is always grave. Many cases of complete recovery have, however, been recorded; but all too often, in spite of rigorous and early treatment, the iris becomes completely bound down to the lens capsule, with occlusion of the pupil, raised tension, and

final phthisis bulbi. Recurrences of inflammation are fairly common, and each attack renders the vision permanently worse.

Prevention and Treatment.—The *exciting eye* should be carefully treated and rendered aseptic, and every effort made to allay inflammation in this, as well as to prevent irritation or inflammation in that of the opposite side. The patient must be warned of the possible danger to the opposite eye, which should be shaded from light, and kept at rest, not only during the attack, but for several weeks after the inflammation of the exciting eye has apparently subsided.

When the exciting eye is evidently rendered useless, either by the wound or by the consequent inflammation—that is, when its vision has quite gone, or only amounts to perception of light, and when there is no probability that its sight will improve—it should be immediately excised. The necessity for its immediate removal is still more urgent when it is giving rise to irritation or to inflammation in the opposite eye.

When the exciting eye, although damaged, is still in the possession of useful vision, or if there is hope of such being restored to it, the question as to whether it should be removed or not becomes most difficult to decide.

If the sympathising eye is suffering from irritation only, and presents no symptom of inflammation, the removal of the exciting eye is usually attended by immediate relief of the irritation, and no symptoms of inflammation are likely to appear; whilst, as we have just seen, the danger of inflammation is very great if the eye is not removed. It therefore becomes a most important and urgent matter to decide whether it is not better to sustain the loss of the eye which is already partially disabled, than to incur the risk of loss of sight to the sympathising eye. Careful consideration of the bearings of the case is necessary in accidents of this kind, which in practice are liable to occur at any moment. A decision having been arrived at, the patient and his friends should be clearly and forcibly warned of the danger to which the sympathising eye is exposed by further retention of the injured eye.

If sympathetic inflammation has already commenced in the second eye, although it may only be of the serous type, then the exciting eye should be preserved; its removal at this late

period is not likely to stop the disease in the other eye ; in fact, the sympathising eye may become so affected by the progress of the disease that the exciting eye may ultimately prove to possess the better vision of the two. Should, however, the exciting eye be completely blind, it must be at once removed. When it is decided to preserve the exciting eye, this should be carefully treated in the manner recommended for plastic iritis, light being excluded from both eyes.

The *sympathising eye* must be treated in the same way as a case of severe plastic iritis (p. 190), that is, with complete rest, exclusion of light, the alternate application of moist and dry warmth, atropine, and leeches, if necessary. In no case, however, must any operative interference, as iridectomy, be attempted, as the aperture caused by the excision of the iris would immediately become filled up by the same exudation as has been thrown out elsewhere. All measures must be actively and promptly used. Inunctions of mercurial ointment are of great assistance in helping the absorption of the inflammatory products. The bowels must be kept freely open, and profuse sweating produced by hot-air baths is often useful.

Operative treatment of either the exciting or the sympathising eye must not be commenced until all inflammatory symptoms have entirely passed away.

The condition of the sympathising eye after the inflammation has subsided is generally very bad. In the mildest cases there are usually extensive posterior synechiæ, but the pupil may remain sufficiently clear to allow of some useful vision. The fixed position of the iris, however, is likely to lead to future inflammatory trouble in the eye, the risk of which would be diminished by the removal of a portion of the adherent iris by the operation of iridectomy upwards. In other cases, the layer of plastic exudation between the iris and the lens capsule is more excessive, and extends to the area of the pupil, which is quite occluded.

The crystalline lens also is frequently involved, and is found to be more or less opaque. Here, of course, the vision is greatly impaired and may amount to perception of light only. An attempt to restore the sight may be commenced by the performance of iridectomy in the upward direction. The operation

Plate V.

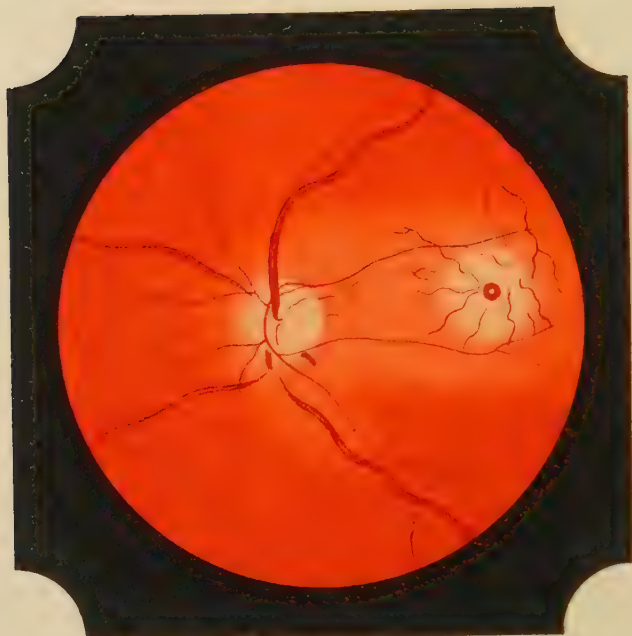


Fig. 1.—Embolism of Central Artery of Retina (*after Liebreich*).

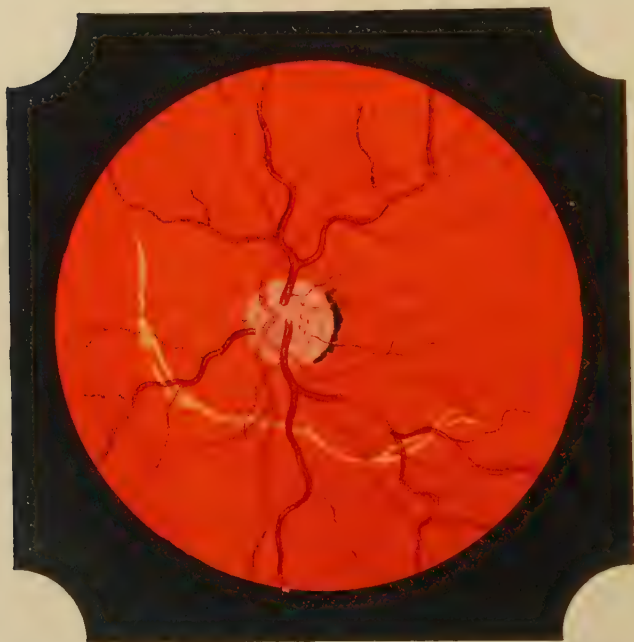


Fig. 2.—Rupture of the Choroid.

is by no means easy to perform, owing to the toughness of the adhesions and the degeneration of the iris tissue. If an artificial pupil can be thus made, and the lens substance is found to be transparent, no further proceeding is at present necessary. Should the lens be found to be opaque, an attempt may be made to extract it through the wound already made for the iridectomy. Its removal is usually attended with difficulty, owing to the extensive iridic adhesions. These may sometimes be more or less detached by means of a Streatfeild's hook ; and even then it is usually necessary to use the scoop in order to get the lens away from its incarcerated capsule. After the recovery from the iridectomy or the extraction of the lens, the vision may sometimes be still more improved by iridotomy (see p. 236).

CHOROIDAL DETACHMENT.

This is a rare condition of the choroid, with, however, a varied etiology. Its commonest cause is a large escape of vitreous during the performance of a cataract extraction or iridectomy. Tumours of the choroid produce choroidal as well as retinal detachment. It may occur during an attack of choroido-retinitis, when the factors which produce it are : (1) choroido-retinitis, producing adhesions between the retina and choroid ; (2) hyalitis with shrinking of the vitreous ; and (3) an exudation of serum behind the choroid. Again, a severe concussion injury may cause detachment of the choroid, as well as of the retina. And lastly, there are the unexplained idiopathic cases. The ophthalmoscopic appearances are very similar to those of retinal detachment alone ; the choroidal stroma may be seen beneath the detached retina and with the same lens, and no motion takes place with the movements of the eyeball. The diagnosis of tumours of the choroid is of extreme importance, and will be discussed under that heading on p. 222. Eyes with a serous detachment usually end in phthisis bulbi. No treatment has as yet met with any success.

TUMOURS OF THE TUNICA VASCULOSA.

Primary sarcoma of the iris is very rare. About half the recorded cases have been melanotic. The diagnosis of the

leucotic variety from tubercle is impossible. The melanotic form may be mistaken for melanoma, which, however, is stationary and non-vascular. Metastasis is common. The eye should be enucleated as early as possible.

Primary sarcoma of the ciliary body is even more rare.

Primary Carcinoma of the Ciliary Body.—A few cases of this disease have been recorded. The cells are pigmented, and are probably derived from those of the ciliary glands.

Sarcoma of the choroid usually commences in a manner so insidious as to be unnoticed even by the patient until the tumour has attained a considerable magnitude; even then it is sometimes discovered accidentally by the patient closing one eye and finding the vision of the affected eye diminished. Sometimes, however, the growth of sarcoma is accompanied by local pains, flashings of light, &c. It usually occurs during middle life or old age, being seldom seen before the age of thirty-five. No cause has been discovered. There appears to be no relationship between it and blows on the eyeball.

Symptoms.—The clinical history of sarcoma of the choroid is usually divided into four stages: (1) the stage of normal tension, with perhaps an initial slightly diminished tension; (2) the stage of glaucoma; (3) the stage of diminished tension, with escape of some of the growth outwards beyond the walls of the eyeball; and (4) the stage of dissemination. When seen at an *early stage*, there may be nothing in the exterior of the eye to attract notice.

In addition to the dimness of sight which may have first caused the patient to apply for advice, we find that the visual field is deficient in some parts, and, when it is examined by means of the perimeter, presents a scotoma corresponding to the position of the tumour. With the ophthalmoscope an outline of the tumour can sometimes be seen to form a rounded prominence, pushing the retina forwards into the vitreous cavity. It is, however, always a matter of difficulty to say whether this is due to a sarcomatous growth in the choroid, or to simple detachment from subretinal effusion. When due to sarcoma, the detached or bulging portion of the retina may retain some colour or be pigmented; it may occur at any part of the fundus, and it does not flap about when the eye is moved.

In old-standing simple detachment, the detached portion has a bluish-white appearance; it usually occurs at the lower segment of the fundus, and it may flap about freely when the eye is moved. Occasionally a vascular network of the sarcomatous growth can be detected through the retina.

Sarcomata in the ciliary region are readily recognised by the changes they produce in the iris. There is at first a bulging forwards of that membrane against the cornea, and eventually it separates from its ciliary attachment, the growth presenting itself through the gap caused by the irido-dialysis. The pupil is altered in shape and is displaced away from the position of the growth. The condition of ciliary sarcoma is usually recognised before it has grown sufficiently large to raise the intra-ocular tension.

In the *advanced stage*, the presence of the tumour is accompanied by a distinct increase of the tension of the globe, and the eye presents other symptoms of glaucoma. The anterior ciliary vessels are congested; the cornea becomes dull in appearance and is more or less deprived of sensation. The anterior chamber gradually becomes shallow by the pressure from behind the iris. The iris is sometimes subacutely inflamed, and forms posterior adhesions to the capsule of the lens, which render the pupil irregular. Not unfrequently the iris is atrophied, and it may be detached at that part of its periphery which corresponds to the position of the tumour. The vitreous also is frequently rendered cloudy by the presence of numerous opacities. The vision has gradually become worse, and is now reduced to bare perception of light. When the disease has progressed to such an extent as to destroy vision, there is frequently considerable trouble from pain in the ciliary region and lachrymation, which are of a more severe character than those met with in true glaucoma.

Examination of the fundus with the ophthalmoscope is now rendered impossible by the opacity of the media; and the increased tension of the globe, together with the general appearance of the eye, frequently renders it a matter of difficulty to decide whether the case be one of sarcoma or true glaucoma. So much is this the case that it occasionally happens that the true state of the eye is not discovered until an operation for

iridectomy has been attempted, and is found to be accompanied by escape of vitreous and by greater hæmorrhage than is usual in glaucoma.

Diagnosis.—Sarcoma of the choroid should always be suspected when an eye that has been gradually losing sight, or been quite blind for some time, is suddenly attacked by pain, congestion, and increased tension, or even if the tension be normal, while the other symptoms exist. In any case of extensive detachment of retina occurring in one eye only, and when there has been no myopia or history of a blow upon the eye, we must be cautious in prognosis. Retinal detachment combined with increased tension, as a rule, denotes a new growth, but the presence of normal or even diminished tension does not exclude the possibility of a tumour. Many cases of choroidal sarcoma have a normal tension, even with an extensive retinal detachment, and especially is this the case if the growth involves the ciliary region. In the very early stages, the tension is frequently diminished. The diagnosis of the earlier stages of intra-ocular growth must depend, therefore, on signs and symptoms other than those of glaucoma. Other causes of retinal detachment must be excluded, and especially injury and high myopia. The position of the detachment is of extreme importance. If due to subretinal fluid, the lowest part of the retina will be detached; if due to growth, it may be detached above, with perhaps a second detachment below. On looking into the eye, should translucency be seen coming from the detached portion, a growth must be suspected. This translucency is due to choroidal blood-vessels which are supplying the neoplasm, and would not be seen if the detachment were simple. On the other hand, a complete fundus reflex excludes a tumour, which would be discovered as a dark area. Other important points in the diagnosis of choroidal sarcoma are: the motionless character of the retinal detachment; the steepness of its edges; the presence of vessels other than the retinal vessels; the presence of pigmentation; the presence of hæmorrhages; and the shadow produced if the whole eyeball be rendered translucent. The diagnosis between sarcoma of the choroid and intra-ocular cysticercus is considered on p. 418. Since increase of tension is a most valuable sign in the

diagnosis of intra-ocular tumours, it is easy to understand how sarcoma of the choroid may be mistaken for *acute primary glaucoma*; and as Hill Griffith¹ points out in his valuable paper, the treatment is diametrically opposite—that is to say, iridectomy is imperatively called for in acute glaucoma, whereas its performance for a new-growth is worse than useless. If the fundus can be seen, there is, as a rule, no difficulty; but supposing the media and cornea are so hazy as to obstruct all view of the fundus, as is not unfrequently the case, the diagnosis must be made by the history of the case, whether any symptoms of glaucoma had previously existed, and by the condition of the visual field and the projection of light. If the vision is completely lost and pain is persistent, removal of the eyeball is the correct treatment, whether it be a case of simple or secondary glaucoma.

Pathology.—Sarcoma of the choroid is usually more or less pigmented (melanotic) and consists of spindle-shaped and round cells. It may be divided into the following varieties:

- | | | |
|--------------------------|---|---|
| A. Leuco-sarcoma | { | (α) Spindle-celled.
(β) Round-celled.
(γ) Mixed-celled. |
| B. Melano-sarcoma | { | (α) Spindle-celled.
(β) Round-celled.
(γ) Mixed-celled. |
| C. Leuco-melano-sarcoma. | | |

Melano-sarcomata are more common than leuco-sarcomata. Of the melanotic variety, the spindle-celled variety is much commoner than the round-celled. On the other hand, round-celled leuco-sarcomata are more common than the spindle-shaped variety. These spindle cells are about $\frac{8}{1000}$ inch in diameter. They contain a large nucleus, surrounded by a hyaline substance (protoplasm), which tapers off at each end. The tumours are usually of firm consistence; they generally contain some blood-vessels, and sometimes are very vascular; the walls of the blood-vessels are composed of sarcomatous elements, as is seen in sarcomata in other parts of the body. They usually bulge towards the vitreous cavity, where the

¹ *Medical Chronicle*, April and May, 1892.

retina is seen to be pushed forwards in front of the tumour. They have an even and smooth convex surface, so long as the lamina vitrea remains intact. When they perforate this membrane it grows more rapidly, becomes hourglass-shaped, and presents an irregular granular surface. Sometimes there is also effusion of serum or blood beneath the retina. They may increase so as to fill the whole globe and distend its walls before invading the extra-ocular tissues of the orbit; but in many cases the tissues outside the sclerotic are affected by the new-growth, whilst the tumour within the globe is quite small; in these cases the cells pass to the outside by means of the sheaths of the blood-vessels, which are seen to be thickened and altered by the presence of cells similar in character to those of the tumour.

The state of tension of the globe is of importance, as it helps us to form some idea of the progress which the new-growth may have made. Thus, if the tension has steadily increased to + 1, + 2, or + 3, we infer that the sclerotic coat is still unaffected. If tension is reduced from + 1, + 2, or + 3 to normal, this indicates that there may be thinning of this tunic. Should the tension be diminished to - 1, - 2, or - 3, we know that the sclerotic has given way in one or more places, and so offers no further resistance to the intra-ocular fluids.

The neighbouring lymphatic glands are not affected, but secondary sarcoma is liable to be set up in distant parts of the body, the cells being conducted from this primary source by means of the blood-current. The liver is the organ which is usually first affected in this way.

The only intra-ocular tumour for which it might be mistaken is that of glioma, which we shall see occurs only in young children.

Course and prognosis.—If left alone, the disease usually takes some years to run through all its stages, the end being always fatal, the patient dying generally from extension into the brain, or from metastasis. In all cases, however early the disease is diagnosed, the eye is lost, and it is impossible to promise that there will be no recurrence, either local or general.

The *treatment* consists in the enucleation of the eye as soon as the disease is recognised. When there is any doubt

as to the presence of a growth, the post-retinal space should be tapped by a posterior sclerotomy, in order that the nature of the retinal detachment may be ascertained ; while absence of fluid indicates the existence of a growth, its presence does not exclude one. The combination of retinal detachment with glaucomatous symptoms and loss of vision is an almost invariable indication for enucleation.

In removing the eye, it is well to take away 4 to 5 mm. of the optic nerve, and to examine the cut end of this after removal. If it should be found to be pigmented or thickened, as much of the remaining nerve as possible should be removed ; in fact, the orbit should be emptied of all its contents. In addition, it is well to remove both lids, as these, if left, are a constant annoyance to the patient, entropion being produced. For an account of the operation see p. 621.

Carcinoma of the choroid is very rare, and is always secondary, usually to carcinoma of the breast. It resembles, in its symptoms, choroidal sarcoma. The eye should not be enucleated until glaucomatous symptoms arise.

Cases of **Melanoma** and **Nævus** of the iris have been recorded.

CYSTS OF THE IRIS.

Cysts of the iris are of three varieties, all of which are rare.

(1) *Epithelioid cysts* are probably always implantation cysts ; a hair in some cases, corneal epithelium in others, being carried by a perforating injury into the iridic stroma. They are lined by squamous epithelium, and contain epithelial débris. They appear some months or even years after the injury, grow slowly, press on the cornea, producing opacity, and encroach upon the pupil, causing loss of sight. Ultimately glaucoma may be set up. When small they should be removed by an iridectomy. If not seen till late, evisceration or enucleation may be necessary.

(2) *Serous cysts* differ pathologically from epithelioid cysts. Their mode of origin is not definitely known. Some suppose that they are formed from the cysts on the anterior surface of the iris, the outlets being occluded by chronic inflammatory exudation. Others consider their formation secondary to

synechiæ, either anterior or posterior. Microscopically, they are lined by a single layer of epithelium. Their course, symptoms, and treatment are similar to those of epithelioid cysts.

(3) Cysts have been described as occurring between the two layers of the uvea. Iritis usually supervenes.

CONGENITAL AFFECTIONS OF THE TUNICA VASCULOSA.

Irideremia or **Aniridia**, or congenital absence of the iris, is occasionally met with. Probably in all cases the iris is not altogether absent, but is represented only by a mere rudimentary band of tissue, occupying the periphery of the anterior chamber. This affection is often accompanied by other defects of the eye, such as partial dislocation of the lens, cataract, choroidal coloboma, nystagmus, and imperfect power of accommodation. It is frequently found in members of the same family.

Coloboma iridis consists in a congenital cleft in the iris. It is usually directed downwards, or downwards and slightly inwards, and resembles the gap which is made by iridectomy, except that the constrictor muscle can be seen to line its walls. It varies in extent, and usually occurs in both eyes. It is often accompanied by coloboma of the choroid. It is frequently hereditary, and has been traced through four generations. It has been produced artificially in the offspring of animals, a small sector of whose irides has been made to become atrophic. Most cases of downward coloboma are probably due to late closure of the choroidal fissure. On account of this there is a cleft in the ciliary body, and consequently here either no iris is developed or it is rudimentary. This, however, does not explain the condition of upward coloboma, which occasionally occurs, nor of a double coloboma in the same eye, an abnormality which has been described. These cases are probably due to some irregularity in the production of the secondary optic vesicle, hindering the development of a sector of the iris.

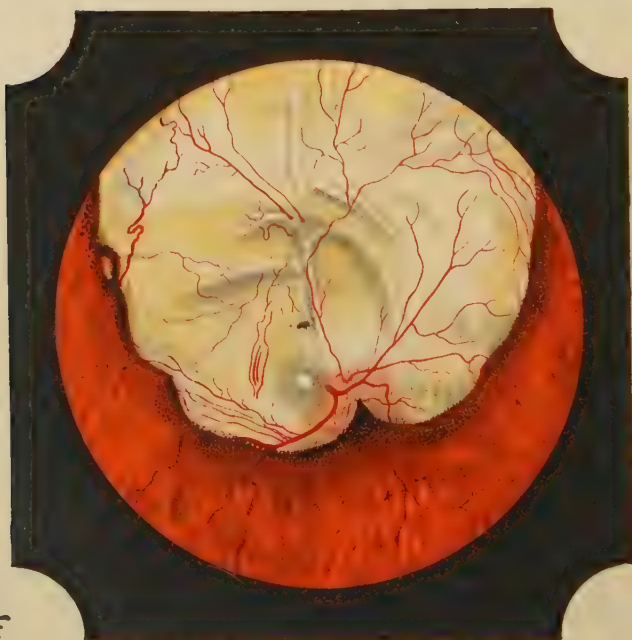
Persistent remains of the pupillary membrane are sometimes found. This membrane is a foetal structure, which closes the aperture of the pupil. Normally it disappears before birth. When persistent, it usually appears as one or

Plate VI.



W.J.R.

Fig. 1.—Congenital Crescent.



W.J.R.

Fig. 2.—Coloboma of Choroid (by indirect method).

more very slender threads extending across the pupil, arising from the anterior surface of the iris, in the neighbourhood of the *circulus arteriosus iridis minor*, and attached either to the opposite portion of the iris or to the anterior capsule of the lens. It can be best seen by the oblique focal illumination. Occasionally a definite membrane is seen in front of the lens.

Iridodonesis (or 'trembling' iris) is found in cases of congenitally dislocated lens.

Coloboma of the choroid is a congenital deformity, which consists in the absence of a more or less considerable portion of this part of the *tunica vasculosa*, and is usually found in the lower and internal part of the globe. When examined by means of the ophthalmoscope, it appears of a bluish-white colour, with clear-cut pigmented edges; a few small vessels are seen scattered over its area, and masses of pigment, varying in quantity, are sometimes present. The surface of the sclerotic often appears very irregular. The extent of the coloboma is variable; it usually extends from the edge of the optic disc nearly as far as the ciliary body. It may embrace the optic disc, in which case the latter is changed in appearance, and looks as if it were hyperæmic by contrast with the white area round it. It may occur in the yellow spot region—*macular coloboma*; it may be very localised, normal choroid being seen all round it; it is often accompanied by a coloboma of the iris. The retina is often involved in the coloboma, though it may be present over the whole site. Occasionally it occurs in both eyes, but when unilateral the left eye is most commonly affected. There is a large scotoma in the visual field, corresponding to the extent of the coloboma, but otherwise the sight in some cases is fairly good. The scotoma is not necessarily absolute, as the retina may be present.

The cause of the defect is probably the formation of adhesions between the developing retina and the mesoblast, the latter of which is not differentiated into choroid and sclerotic. This usually takes place in the position of the retinal fissure, and thus accounts for the usual position of the coloboma. It may, however, take place in any part of the retina; hence the occasional occurrence of upward or lateral coloboma of the choroid.

Polycoria is a peculiar condition in which the natural pupil

is divided into two or more apertures by bands of iridic tissue. It is an exceedingly rare condition. Sometimes the extra pupil is situated at the periphery. As many as eight extra pupils may occur, each surrounded by muscular tissue, as shown by its behaviour to atropine.

Heterochromia (heterophthalmos) is a congenital colour peculiarity of the irides. It may be partial or complete. Partial heterochromia is that condition in which a segment of the iris differs in colour from the rest of the membrane; it may be symmetrical. Complete heterochromia is when the iris of one eye is of a different hue from that of the other; the colour of the father's irides will correspond to one, and that of the mother's to the other. Small dots of pigment on the iris may be mistaken for foreign bodies; they are usually, however, multiple and symmetrical.

Occasionally small masses of pigment are seen at the pupillary margin; these are extensions of the uveal pigment of the iris, and are found congenitally, as well as in chronic glaucoma. It is normally found in the horse, and is termed *ectropion of the uveal pigment*.

Albinism, or congenital absence of pigment throughout the body, may be relative or absolute. In *absolute* albinism the pupils and irides appear pink from the reflections of the choroid, the irides also on account of the blood contained in their vessels. This condition is usually attended with defective visual acuity, photophobia, and nystagmus. Upon ophthalmoscopic examination, the choroidal vessels are seen most distinctly as a pink fenestrated membrane upon a pale, almost white, background. The hair is usually white throughout the body. In *relative* albinism the hair is of a pale straw colour; the irides present a pale purplish hue, and do not completely shut off the choroidal reflex; the symptoms also are less marked. There is a tendency to acquire pigment as the child grows; the improvement, however, is rarely more than a change from the absolute to the relative condition. By way of treatment, any existing error of refraction must be corrected, dark glasses should be worn, with or without a diaphragm to cut off some of the light. Peripheral tattooing of the cornea has been performed for the same object.

Congenital crescent is a peculiar greyish-white crescentic patch immediately below or, in rarer instances, to the outer side of the optic disc. The disc, excluding the crescent, is usually oval, but with it appears circular or slightly oval in the opposite direction. These crescents present no marginal pigment, which is so often seen in myopic crescents (compare fig. 1, opposite p. 227, with fig. 1, opposite p. 203); besides, they differ from the latter in being usually below the disc. It is very probable that this crescent is a partial coloboma of the choroid.

Corectopia, or eccentric pupil. The pupil may be congenitally misplaced—so much so that it may be situated quite at the periphery, close to the sclero-corneal junction. It is a rare deformity, and is frequently accompanied by dislocation of the lens.

OPERATIONS ON THE IRIS.

Iridectomy consists in the excision of a portion of the iris. This operation, as is mentioned under the different headings, is frequently performed in various affections of the eye; it forms the preliminary stage of some of the operations for the extraction of cataract; it constitutes a prominent feature in the treatment of glaucoma; it is occasionally resorted to in purulent infiltration, and in certain forms of ulcer of the cornea; it is adopted, with great benefit, in many cases of chronic and recurrent iritis, of irido-cyclitis, of irido-choroiditis, and of anterior staphyloma.

Iridectomy is also resorted to in the majority of cases where an artificial pupil is required for *optical* purposes, as in central opacities of the cornea, which cover up the front of the pupil, and so prevent vision. Also in some forms of cataract, as the stationary variety of lamellar and the anterior pyramidal, which are sufficiently clear at the peripheral zone to admit of distinct vision, after an artificial pupil has been made.

The instruments required for iridectomy are: (1) speculum, fig. 42; (2) fixation forceps, fig. 43; (3) either a bent triangular keratome, fig. 48, or Graefe's cataract knife, fig. 49, or a bent broad needle, fig. 56; (4) a pair of straight or curved

iris forceps, fig. 51, or a Tyrrel's hook, fig. 58; (5) a pair of iris scissors, fig. 50, 52, or 53; and (6) a curette, fig. 91.

The operation varies in detail according to the object with which it is performed. It is divided into three stages: the first stage



FIG. 48.—Bent Triangular Keratome.



FULL SIZE

FIG. 49.—Graefe's Cataract Knife.

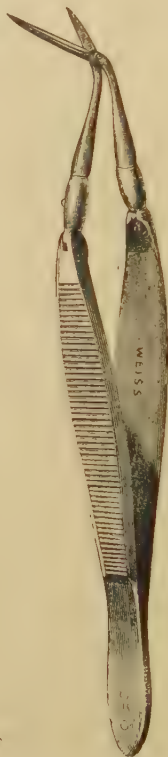


FIG. 50.—Weiss's Iris Scissors.



FULL SIZE

FIG. 51.
Iris Forceps.

consists in opening the anterior chamber by an incision close to, or at, the sclero-corneal junction; the second in seizing, drawing out, and excising the portion of iris to be removed; the third in the 'toilet' of the wound. When the operation is intended for the

relief of glaucoma, or for the purpose of subduing or preventing inflammatory affections, the iridectomy should be made in the upward direction, so that the coloboma thus formed in the iris shall be situated beneath the upper eyelid. In most cases the local anæsthetic action of cocaine will be sufficient, though sometimes,



FIG. 52.—Iris Scissors.



FIG. 53.—Iris Scissors.

as in acute primary glaucoma, a general anæsthetic will be necessary. The patient must lie upon a firm table or couch, of such a height that his head reaches the level of the umbilical region of the operator, who stands behind the patient's head. The eyelids are

kept open by means of a spring-top speculum (fig. 42), and the globe is held steady by seizing the conjunctiva and subconjunctival tissue with fixation forceps (fig. 43) at the part immediately

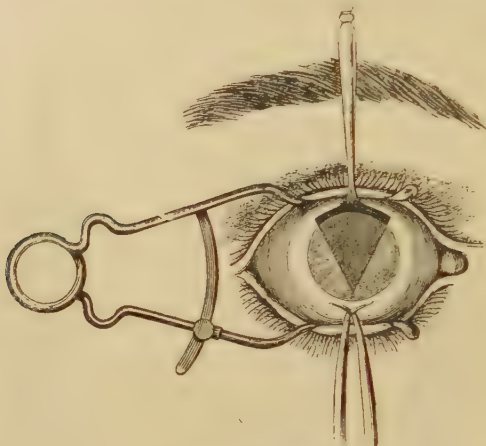


FIG. 54.—Iridectomy with Keratome.

opposite to that at which the incision is about to be made. A bent triangular keratome (fig. 48) is then deliberately inserted at the sclero-corneal junction, and pushed downwards across the anterior chamber until the wound thus made is from 6 to 8 mm. wide (see

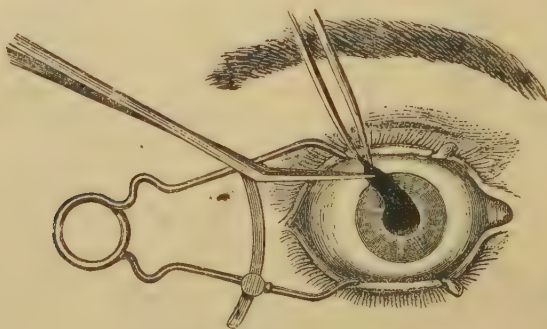


FIG. 55.—The Iridectomy.

fig. 54). In doing this, the instrument is passed in obliquely and in such a direction that if continued it would cause a wound of the iris and lens; as soon, therefore, as its point is seen through the

clear cornea, the handle is slightly depressed, so as to bring the blade into a plane anterior and parallel to that of the iris (fig. 54).

The keratome is now steadily withdrawn. In doing this it is important to keep its apex well away from the plane of the iris and lens. Its withdrawal is accompanied by an escape of the aqueous, which may cause protrusion of the iris between the lips of the wound.

The keratome is now laid aside, and the fixation forceps entrusted to an assistant, who, if necessary, holds the globe in a state of slight rotation downwards, but *without undue pressure or traction*. The closed iris forceps (fig. 51) is now passed into the anterior

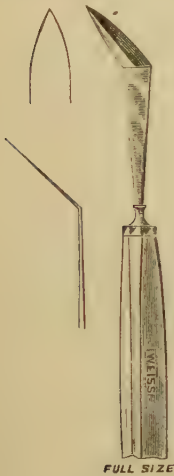


FIG. 56.—Bent Broad Needle.

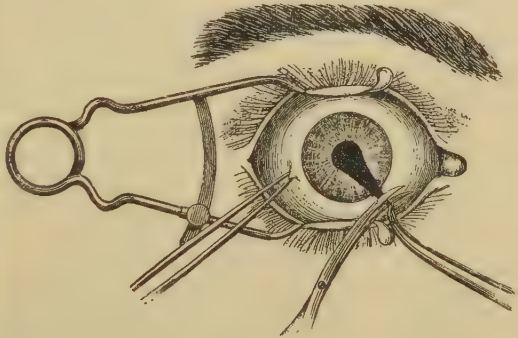


FIG. 57.—Iridectomy for Artificial Pupil.

chamber, the iris is seized near its pupillary edge, and dragged just outside the wound; whilst slight traction is made upon it in this position, the projecting portion is cut off with one snip of the scissors. Should, however, a large iridectomy be indicated, the iris is dragged just outside one angle of the wound, and a snip made through its outer part in the manner shown in fig. 55; the portion of iris held in the forceps is then gently drawn across to the other angle, and the excision completed as near to its periphery as possible. Finally, the curette should be passed into the angles of the wound, so as to liberate any portion of iris that is entangled there, and the edges of the wound are brought into exact apposition. The speculum is then removed, the eyelids are gently closed, and a small

light pad of aseptic gamgee tissue applied either by adhesive plaster or a bandage. Both eyes should be covered.

When the anterior chamber is very shallow, by the bulging forwards of the iris and crystalline lens, the danger of wounding the latter is lessened by the use of the linear knife (fig. 49), as in the preliminary iridectomy for cataract extraction (see Cataract). Some surgeons, however, always make use of the linear knife in performing iridectomy.

When the operation is required for optical purposes only, the position of the new pupil necessarily depends upon that of the lesion of the cornea. The best position, when possible, is either downwards and slightly inwards, or straight downwards. The object here is not to remove a large portion of the iris, but only so much as is necessary for distinct vision. Instead of the large bent keratome, a narrower one (broad needle, fig. 56) is employed for the first stage of the operation. The position of the first incision must depend upon the situation in which the new pupil is required. When this is only slightly eccentric, the incision can be made just within the margin of the cornea. When the pupil is required to be opposite the margin of the cornea, the incision must be made at the sclero-corneal junction. The width of the incision should in either case be at least 3 mm. The globe is fixed by the assistant. The iris forceps (fig. 51) is now passed into the anterior chamber, and the iris seized at its pupillary edge and gently withdrawn through the wound, and, whilst held in this position, the portion which is outside the wound is snipped off close to the globe with the iris scissors (see fig. 57).

When the pupil is required to extend quite to the margin of the cornea, slight pressure should be made upon the globe with the scissors as the iris is being cut away.

Instead of the iris forceps, a Tyrrel's hook (fig. 58) may be used. This is introduced on the flat, and passed as far as the centre of the pupil; it is then half rotated downwards, so as to catch the pupillary edge of the iris, then once more turned on the flat and withdrawn, by which means the iris is extracted, and cut off as before.

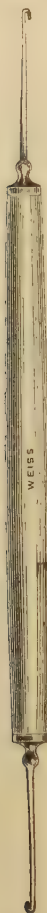


FIG. 58.—Tyrrel's
Hook for Irid-
ectomy.

The accidents and complications of iridectomy.—1. The lens may be wounded during either the insertion or the withdrawal of the keratome. This is a very serious accident, as it is sure to be followed by partial or complete cataract.

2. The blade of the keratome may get between the layers of the cornea, instead of passing directly into the anterior chamber. This accident arises from it being held too obliquely at the *commencement* of the incision. As soon as this condition is discovered, the instrument should be immediately withdrawn, and another position selected for a fresh incision; if, however, the blade has finally entered the anterior chamber, so as to cause escape of the aqueous, the eye had better be bandaged up at once, and the operation postponed for at least twenty-four hours, in order that a re-secretion of aqueous may take place before the knife is again allowed to enter the anterior chamber. Without this precaution, the iris and lens are so pushed forward after the escape of aqueous that they are sure to be wounded at the time of the fresh incision.

3. When the incision is made in the sclerotic, there may be considerable hæmorrhage into the anterior chamber either before or after the excision of the iris. The blood can usually be made to flow out by depressing the upper lip of the wound with the curette. When the excision of the iris is completed, no anxiety need be entertained on account of the presence of a moderate amount of blood in the anterior chamber, as it usually becomes absorbed within a few days.

Iridodesis (G. Critchett) consists in drawing the pupillary edge of the iris through a small opening in the margin of the cornea, and securing it by a fine silk ligature on the outside.

The incision is made immediately in front of the sclero-corneal junction, by means of a broad needle, bent at an obtuse angle. The needle is then removed, and a loop of fine black silk is placed immediately around the wound. A Tyrrel's hook or an iris forceps is then passed through the loop and wound into the anterior chamber, and the pupillary edge of the iris seized and withdrawn through the wound to the desired extent. Whilst the iris is held in this position by the operator, the two ends of the ligature are picked up by the assistant, by means of a broad cilia forceps; they are then tightened close to the surface of the cornea, and the knot is completed. The strangulated portion of iris quickly shrinks, and the ligature can be removed after a few days. By this means the original pupil is shifted to one side, and a new somewhat pear-shaped pupil is formed.

This method is particularly useful in certain cases of conical cornea and lamellar cataract, also where a central nebula of the cornea is sufficient to blur the vision, though not to exclude the light; the original pupil being obliterated by the traction upon the iris, the rays which formerly passed through the nebula are now excluded, whilst the new pupil admits only those rays which pass through the clear part of the cornea. Iridodesis was formerly practised somewhat extensively by G. Critchett, Bowman, and others; but the occurrence of sympathetic irritation and of sympathetic ophthalmitis in a few cases where the operation had been performed has caused it to be less frequently adopted.

Iridotomy (iritomy) consists in the formation of an artificial pupil by simple incision of the iris. It can only be safely adopted when the crystalline lens is absent, and is mostly applied to those cases in which the iris has become tightly drawn upwards towards the cicatrix as the result of inflammation after the extraction of cataract.

Operation.—The eyelids being separated, and the globe steadied, as in the previous operations, a narrow lance-shaped keratome (fig. 56) is plunged through the upper part of the cornea about 2 mm. from the sclero-corneal junction; it is then pushed onwards through the membranous exudation to the back of the iris, and finally withdrawn. The iridotomy scissors (fig. 50) are now passed through the corneal wound, their blades being closed; as soon as they reach the iris, one blade is passed behind and the other in front of that structure, which is now divided by a single snip from above downwards; this single incision is usually followed by immediate separation of the cut edges so as to form a slitlike or triangular pupil. In some cases it is necessary to make a second incision at an acute angle with the first, so as to include a V-shaped piece of iris, which can either be left to atrophy or be removed.

Iridotomy is also practised for the production of artificial pupil in certain cases of lamellar cataract, &c.; the advantage claimed being that the small slitlike aperture thus obtained is better for optical purposes than the larger opening produced by even a small iridectomy.

In such a case the incision in the cornea must be made at the side opposite to that at which the new pupil is required; it should be about

4 mm. wide. De Wecker's or Weiss's iridotomy scissors (fig. 50) are now carefully introduced to the anterior chamber; having reached the pupil, the blunt-ended blade is passed behind the iris, between it and the capsule; the other blade, which is usually gilt, is passed in front of the iris in the direction of the desired pupil; the iris is now divided by a single cut, and the closed instrument is cautiously withdrawn.

PRIMARY DEGENERATIONS OF THE CHOROID.

There are many rare ophthalmoscopic appearances of the choroid, which are extremely difficult to classify, as they overlap each other to a considerable extent, and as their pathology is still very obscure.

Guttate choroiditis of Tay is characterised by a number of yellowish-white specks which are generally, though not necessarily, confined to the macular region. They are usually found in those who are past middle life. Vision is little, if at all, affected, and the progress of the disease is extremely slow. It is probably a punctate colloidal degeneration of the lamina vitrea of the choroid.

Colloidal degeneration in the young is occasionally found. It differs from Tay's guttate choroiditis in not being confined to the macular region. The visual acuity and visual field are normal, and night-blindness is absent.

Retinitis punctata albescens of Nettleship is a primary degeneration of the retina and choroid (see p. 294).

Atrophia gyrata choroideæ et retinae of Fuchs is a rare form of atrophy (see p. 294).

Ossification of the choroid frequently follows *phthisis bulbi*. It may be extensive, a thin plate being present in the inner or vitreous part of the choroid; or there may be merely a few spicules of bone scattered throughout the choroid.

CHAPTER VII.

THE OPTIC NERVE AND RETINA.

ANATOMY AND PHYSIOLOGY OF THE OPTIC NERVE—OPHTHALMOSCOPIC APPEARANCE OF THE NORMAL OPTIC DISC—ANATOMY OF THE RETINA—PHYSIOLOGY OF THE RETINA—OPHTHALMOSCOPIC APPEARANCE OF THE NORMAL RETINA—PULSATION OF THE RETINAL VESSELS—CONGENITAL ANOMALIES—HYPERÆMIA OF THE OPTIC DISC—OPTIC NEURITIS—OPTIC ATROPHY—HÆMORRHAGES INTO THE OPTIC-NERVE SHEATH—DISEASES OF THE RETINAL VESSELS—RETINAL ISCHÆMIA—EMBOLISM OF THE CENTRAL ARTERY—SYMMETRICAL INFANTILE MACULAR CHANGES—THROMBOSIS OF THE CENTRAL ARTERY—THROMBOSIS OF THE CENTRAL VEIN—RETINAL HÆMORRHAGES—THE RETINA IN GENERAL ARTERIO-SCLEROSIS—ANGIOID STREAKS IN THE RETINA—RETINITIS—ALBUMINURIC RETINITIS—DIABETIC RETINITIS—LEUCOCYTHEMIC RETINITIS—SYPHILITIC RETINITIS—RETINITIS CIRCINATA—RETINITIS PROLIFERANS—DEGENERATIVE CONDITIONS OF THE RETINA—PIGMENTARY RETINITIS—RETINITIS PUNCTATA ALBESCENS—DETACHMENT OF THE RETINA—TUMOURS OF THE OPTIC NERVE AND RETINA—GLIOMA OF THE RETINA.

ANATOMY AND PHYSIOLOGY.

The Optic Nerve.—Each optic tract arises by two roots, of which the *external* (anterior brachium) takes origin from three centres of grey matter—viz. the pulvinar of the optic thalamus, the external geniculate body, and the anterior tubercles (nates) of the corpora quadrigemina—the *three primary visual centres* (Foster); while the *internal* (posterior brachium) arises from the internal geniculate body and the anterior and posterior tubercles (testes) of the corpora quadrigemina. The fibres which form the posterior brachium resolve into the inferior commissure of Gudden and the fibres of Meynert; they are not true visual fibres. The true optic centres are connected with the cerebral cortex by a system of fibres—the *visual tract*—which pass upwards through the internal capsule in the hinder part of the main sensory tract and spread out in the *optic radiation* to the cortex of the occipital lobe.

Its exact cortical centre has not at present been accurately determined, though it appears highly probable from recent *post-mortem*

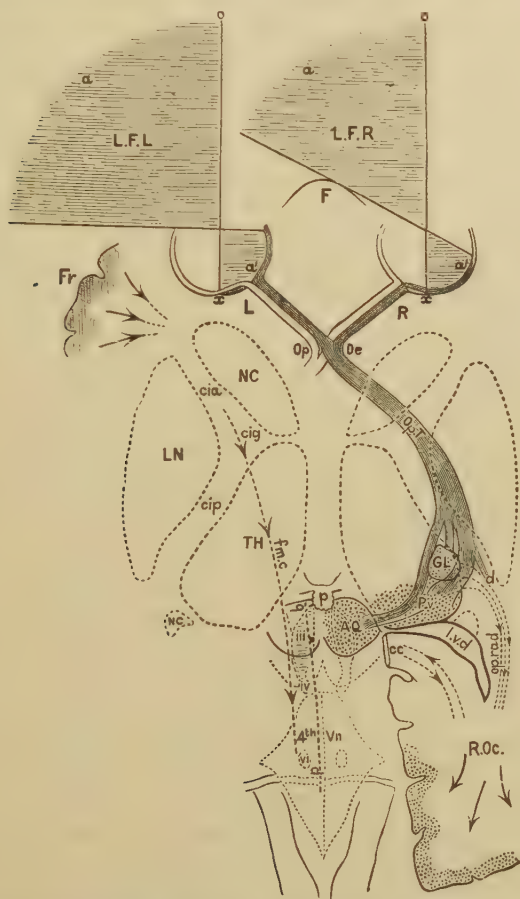


FIG. 59.—Diagram to illustrate the Nervous Apparatus of Vision.
(Sherrington.)

L, left eye; R, right eye; o.x., optic axis; F, outline of face between the eyes; Op. T., right optic tract (shaded); Op. De., optic decussation; L.F.L. and L.F.R., the left visual fields of the left and right eye respectively; G.L., corpus geniculatum externum; Pv, the pulvinar, and AQ, corpus quadrigeminum anterius (all stippled); op. rad., optic radiation; R.Oc., the right occipital lobe, whose stippled cortex indicates the 'visual area'; d, the direct tract of Wernicke to cortex; cc, corpus callosum; l.v.d., descending horn of lateral ventricle; Fr indicates the cortical motor area for the eyes; fm.c., the motor path from the cortex to the nuclei of the third, fourth, and sixth nerve-nuclei, iii, iv, vi.

examinations in cases of hemianopsia that it is situated in the cuneus and superior occipito-temporal convolution—that is to say,

at the apex of the occipital lobe, and probably more on the mesial than the external aspect of the hemisphere. A few fibres of the optic tract are said to pass directly to the cortex through the posterior part of the internal capsule, without the interruption of basal ganglia (direct tract of Wernicke). In the anterior corpora quadrigemina, fibres from both tracts probably come into connection with the nuclei of the ocular muscles. Besides this occipital, or *perceptive*, centre, there appears to be a higher, or *memory*, centre, situated in the left angular and supramarginal convolutions of the parietal lobe in right-handed individuals, and on the opposite side of the brain in left-handed people. This centre is connected with both of the occipital centres. The *optic tract* formed by the union of its two roots then passes forwards along the posterior inferior surface of the optic thalamus, winds round the crus cerebri, and runs along the side of the tuber cinereum to the front of the infundibulum, where it unites with the optic tract of the opposite side to form the *optic commissure* or *chiasma*. While crossing the grey matter forming the floor of the third ventricle it gives off the fibres of Meynert, and at the chiasma the rest of the fibres of the posterior brachium arch back along the opposite tract to the internal geniculate body of the other side. The fibres from the anterior brachium undergo semi-decussation: the greater part passes to the *nasal* half of the retina of the *opposite* eye, the rest supply the *temporal* half of the eye *on the same side* (fig. 59). This direct bundle is thought by some to undergo decussation in the anterior corpora quadrigemina; there is, however, no definite anatomical evidence of this. The fibres coming from the central part of the retina, the papillo-macular bundles, divide at the chiasma into a crossed nasal bundle and a direct temporal bundle. From the optic commissure the two optic nerves arise, and pass forwards and outwards to the two optic foramina. As they pass through these, they become invested by prolongations from the pia mater, the arachnoid, and the dura mater of the brain, which, unlike the rest of the cranial nerves, remain distinct. To the optic sheath at the optic foramen are attached the capsule of Tenon, and the origins of the superior and the internal recti muscles. Each nerve is about 4 mm. in diameter, and its orbital portion is about 28 mm. in length. It has an S-shaped passage through the orbit to allow of the ocular movements, as well as permitting exophthalmos. It is completely surrounded by orbital fat, and passes forwards, outwards, and slightly downwards. As it enters the orbit it lies above and to the inner side of the ophthalmic artery. Later it is crossed by this vessel from without inwards, together with the companion veins and the

nasal nerve. The nerve is made up of numerous parallel bundles of nerve-fibres, with intervening septa of connective tissue derived from the pial sheath. The individual fibres of adjoining bundles anastomose with each other. The nerve-fibres possess the medullary sheath or the white substance of Schwann, but are devoid of the external primitive sheath, or neurilemma; the axis cylinders with their fatty insulation are supported by a very delicate network of fine connective tissue (neuroglia). The ophthalmic artery entirely supplies the optic nerve with blood. In front of the lamina cribrosa a fine network of vessels is formed by the short posterior ciliary branches—the *circle of Zinn*. Minute and occasionally large branches from the arteria centralis retinae take part in this anastomosis, which nourishes the anterior part of the nerve. Sometimes branches, the cilio-retinal, pass to the retina. It is still a disputed question whether the optic nerve is entirely composed of afferent nerves. Von Monakow considers the large fibres to be afferent, and the small fibres, fewer in number, to be efferent, arising from a centre in the mid-brain and ending in the inner granular layer of the retina.

Near the globe (about 15 mm. behind it) the optic nerve is penetrated in its inferior nasal quadrant by the central artery of the retina with its vein. These are accompanied by distinct lymphatic sheaths, and pass obliquely to the centre of the nerve. The central artery of the retina does not supply the optic nerve with blood, though it gives off a few minute branches immediately behind the lamina cribrosa, which pass forward in a parallel direction to supply that structure and the optic papilla.

The *pial sheath* is a fibro-vascular structure, which closely invests the nerve, and sends off numerous bands between the fibres of the latter, so as to form a network of trabecular tissue; the trabeculae thus formed give off still finer connective-tissue filaments, which extend between the nerve-fibres. The pial sheath terminates anteriorly by blending with the inner fibres of the sclerotic at the edge of the optic disc, and partly with the choroid. It is supplied with blood from the branches of the ophthalmic artery, and thus, by its continuity with the pia mater, establishes arterial communication between the intracranial and orbital arteries.

The *dural sheath* forms a complete fibrous investment to the nerve, and terminates anteriorly by blending with the sclerotic at the optic nerve entrance.

The *intersheath* or *intervaginal space* is the space between these two membranes, and is considered to be a lymph space (Schwalbe). It is imperfectly divided by a delicate prolongation of connective tissue from the *arachnoid membrane* of the brain; this is attached

partly to the dural and partly to the pial sheath, and subdivides the space into the subdural space externally and the subarachnoid internally. The intersheath space communicates posteriorly with the subdural space of the brain, and anteriorly with certain lymph spaces in the optic nerve at the lamina cribrosa. The arachnoid membrane fuses with the dural sheath close to the eyeball.

The fibres of each optic nerve are arranged in such a way that those coming from the peripheral parts of the retina occupy a central position, while those coming from the central parts of the retina are placed in a marginal position. The so-called papillo-macular bundle, however, which occupies the part of the retina lying between the papilla and the macula, is an exception to this rule. It first forms a sector in the temporal quadrant of the nerve; later it becomes central; and at the optic chiasma it occupies the dorsal half.

The *optic disc* or *papilla*, the ocular termination of the optic nerve, is formed by the radiating fibres of the optic nerve immediately after their passage through the openings in the sclerotic and choroid at the back of the eye. It is 1.6 mm. in diameter, and is 15° (or 3 mm.) to the nasal side of, and a little higher than, the posterior pole of the eyeball; usually circular, it may be slightly oval, the longest diameter being vertical. It is placed in the inferior nasal quadrant. The *sclerotic opening*, as we have just seen, is guarded by the lamina cribrosa. In front of this is a delicate layer of connective tissue containing capillaries, which is derived from the choroid. The *capillaries* of the disc are supplied from three sources—viz. the posterior short ciliary arteries of the choroid, the central artery of the retina, and the arterial twigs of the pial sheath. These three sets of vessels anastomose freely at the optic disc. As the nerve-fibres pass through the lamina cribrosa they become divested of their medullary sheaths (white substance of Schwann), and are reduced to axis cylinders only, surrounded by a little transparent gelatinous substance (neuroglia); the whole nerve in consequence becomes considerably contracted. Being thus rendered quite transparent, they radiate towards the retina.

The Ophthalmoscopic Appearance of the Optic Nerve.—When examined with the ophthalmoscope, the healthy optic disc usually appears as a whitish circular area surrounded by the orange-coloured groundwork formed by the choroid. It is not a perfect circle, but is slightly elliptical, its longest axis being vertical. It usually has a slight pinkish tint, such as is presented in figs. 1 and 2, opposite p. 198; but its appearance is subject to numerous slight variations, which can only be learned by frequent examination of healthy fundi. The white reflection is caused chiefly by the lamina

cribrosa, which shines through the transparent nerve-fibres, partly by the white substance of the nerve-sheaths which terminates just behind the disc ; it is usually most marked at the outer part of the disc, where the fibres are thinnest. The pinkish tint is due to the presence of capillaries, and is more marked when these are distended than when they contain but little blood. In fair persons, where the pigment-layer of the retina is thin, the disc often appears darker by contrast with the rest of the fundus than in persons of dark complexion. It occasionally happens that the disc looks quite white, although the visual and other functions of the eye are normal.

The central artery of the retina is seen to emerge from the depths of the optic nerve rather to the inner side of the centre of the disc, and to the nasal side of the vein ; it sometimes divides before traversing the lamina cribrosa, but more commonly its point of bifurcation is anterior to that structure, and can be seen from the front. The two chief divisions thus formed pass vertically, the one upwards and the other downwards, to the retina (see Retina). The central vein accompanies the artery, and is distinguished by its somewhat darker colour and larger size ; it usually enters the papilla as a single trunk, and lies to the temporal side of the artery.

Along the margin of the disc there is often seen a small patch or line of dark pigment. It may occur at any part of the circumference, and is of no pathological importance.

The **retina** is a delicate membrane containing the terminal end-organs of the fibres of the optic nerve, supported by a connecting framework. It lies between the choroid and the hyaline membrane of the vitreous humour, and extends from the optic disc to the outer part of the ciliary processes, where it presents a finely indented border, the *ora serrata*. At this point the differentiated elements of the retina cease, but their representative, a layer of columnar cells, is continued forwards under the name of the *pars ciliaris retinæ* as far as the zonula. The membrane diminishes in thickness from 0.4 mm. posteriorly to 0.2 mm. anteriorly. At the back of the retina is a layer of pigment-cells which is continuous with the pigmentary layers of the iris and ciliary body already described. This layer adheres to the choroid when the latter is separated from the retina ; it was formerly considered to belong to that membrane, but the study of the development of the part shows that it belongs to the retina. By carefully removing the anterior portion of the globe with scissors, and clearing away the vitreous (immediately after an eye has been excised from the living subject), we find that the inner surface of the retina is smooth, and that its substance is quite transparent. About 3 mm. to the inner side of the posterior

pole of the globe is seen a white almost circular disc of about 1.6 mm. diameter. This is the *optic disc* or *papilla* (p. 242), from the centre of which the radiating retinal veins are plainly visible as red streaks. At the posterior pole the brown colour of the pigmentary layer is observed to be intensified over a small area; this is the yellow spot (*macula lutea*), and if the segment of the globe be placed in water and examined under a low power of the microscope, this area will be found to be depressed at the centre; the depression is the *fovea centralis*. In most cases also there will be found a yellowish appearance; hence the name of 'yellow spot' which has been given to this, the most sensitive portion of the retina, although in some cases the difference of colour between this region and the remainder of the retina is extremely slight. The yellow spot region is about 1.25 mm. in diameter, and is somewhat elliptical in shape, the long axis being horizontal.

Microscopic anatomy.—The elementary structures of the retina are arranged in several layers. These may be enumerated from behind forwards, as follows :

- | | |
|-------------------------------|-------------------------------|
| 1. The pigmentary layer. | 5. The inner granular layer. |
| 2. The rods and cones. | 6. The inner molecular layer. |
| 3. The outer granular layer. | 7. The ganglionic layer. |
| 4. The outer molecular layer. | 8. The nerve-fibre layer. |

Besides these structures, the retina contains a connecting framework, which is composed of

1. The outer limiting membrane.
2. The fibres of Müller.
3. The inner limiting membrane.

1. The *pigmentary layer* bounds the retina externally. It is the outer lamina of the optic vesicle, and consists of a single layer of nucleated polygonal cells, which are chiefly six-sided. Some are, however, five-sided, and others may have as many as ten sides. The outer surface of each cell is smooth, flattened, and devoid of pigment, and contains the nucleus. The inner surface is loaded with pigment-granules, and is prolonged by filamentous processes into the region of the rods and cones.

2. The *layer of the rods and cones* is the most important part of the retina. The rods and cones are placed perpendicularly to the plane of the retina. The rods extend externally as far as the pigment-layer, and are cylindrical in form. The cones are shorter, thicker, and bulged at their inner extremity, whilst they terminate externally by a tapering filament which does not reach the pigment-

layer. Both of these structures are divided into two segments—an outer and an inner. The outer rod-segments are cylindrical in shape and fibrillated in structure, and have a remarkable tendency to split up spontaneously into highly refractile superimposed circular discs, presenting the appearance of a pile of coins; they are unaffected by carmine, iodine, and other stains. They are of a purple colour, on account of the visual purple or rhodopsin they contain. The outer cone-segments resemble the outer rod-segments in all particulars, except in their being conical rather than cylindrical, and in possessing no rhodopsin. The inner rod-segments are singly refractile, stain with carmine, and are of a slightly larger diameter than the outer segments, though tapering somewhat at each end. The outer portion is finely fibrillated longitudinally, while the inner is finely granular. The inner cone-segments have a very similar structure. Their diameter is, however, greater, and the outer portion is larger than the inner portion.

3. *The outer granular layer* (outer nuclear).—On entering this layer, each rod almost immediately becomes a fine tapering fibre, which, after passing a variable distance through this layer, expands to enclose an oval transversely striated nucleus, and is continued on as a finely nodulated filament, until it enters the next layer. Each cone, on the other hand, bears its nucleus immediately on entering this layer, and before its diameter is much diminished. Continuing on as a nervous filament, broader however than the rod filament, it can be traced into the outer molecular layer.

4. *The outer molecular layer* contains the endings of the rod and cone fibres. The rod-fibres end in small knoblike expansions, whereas the cone-fibres expand into broad bases or 'feet.' These terminations are each in turn surrounded by a network of fibrils connected with the cells found in the succeeding layer, immediately to be described. Besides these fine fibrillæ, which are the chief component of this layer, a few large so-called 'horizontal cells' are found, the processes of which are chiefly confined to this layer.

5. *The inner granular layer* (inner nuclear) contains two varieties of large nerve-cells which are very similar to the bipolar ganglionic cells, having a large nucleus and a small nucleolus. Each of these cells has two tail-like processes—one passes upwards to the preceding layer, the other, the axis-cylinder process, goes in the opposite direction. One of these varieties of cells is in close contact with the terminations of the rod-fibres, and in such a way that the fibrillæ from the ascending process of one cell surround the endings of several rod-fibres. The axis-cylinder process of this variety passes through the succeeding or inner molecular layer, and

terminates in a brushwork of fibrils surrounding a cell of the ganglionic layer. The ascending process of each of the second variety of cells of this layer similarly surrounds by its branches several of the bases of the cone-fibres; the axis-cylinder processes end in the inner molecular layer, in brushlike expansions opposite similar endings of ascending processes from cells of the ganglionic layer.

But besides these two forms of bipolar cells, this layer of the retina contains many unipolar cells, the *spongioblasts* or *amacrine* cells, which have no axis-cylinder processes. They are placed deeply in this layer, and are of various shapes, but it will be sufficient to state that their single processes end for the most part in horizontal brushes, placed in the inner molecular layer.

6. The *inner molecular layer* is completely free from nerve-cells. It is composed of fine fibrillæ coming from (a) the axis-cylinder processes of the second variety of bipolar cells of the inner granular layer described above; (b) the ascending processes of some of the multipolar cells of the ganglionic layer, immediately to be considered; and (c) the brushlike endings of the amacrine cells.

7. The *ganglionic layer* is composed of a single layer of large multipolar cells containing a nucleus and a bright nucleolus; their prolongations are directed inwards to communicate with the axis cylinders of the nerve-fibre layer, and outwards into the inner molecular layer.

8. The *nerve-fibre layer* is formed by the fibres of the optic nerve on their way to the ganglionic cells; these consist simply of axis cylinders surrounded by a little neuroglia. The fibres form a fine-meshed reticulum, and finally all radiate towards the optic disc. One bundle, however, has a different arrangement. This is the *papillo-macular bundle*, which passes almost in a straight line from the macula to the disc. The exact relationship between the fibres and cells of the various layers of the retina is still under discussion, but it seems probable that contiguity, rather than continuity, is the system employed. The terminations of the rods and cones in the outer molecular layer are in close contact with the ascending processes of the cells of the inner granular layer; while the axis-cylinder processes of these same cells end either in the inner molecular layer in close contact with the ascending processes of the ganglionic cells, or immediately surrounding the cells themselves in the ganglionic layer. The ganglionic cells are, finally, in direct continuity with the optic-nerve fibres.

The *fibres of Müller* act as a connecting framework, riveting, as it were, the various layers of the retina together. They are formed

from modified neuroglial cells, and are epiblastic in origin, thus differing from true connective tissue. Their outer processes are in apposition, and their extremities form, at the junction of the layer of rods and cones with the outer granular layer, a fenestrated membrane, or the *external limiting membrane*, embracing in its meshes the rods and cones. Projecting upwards between the rods and cones are small processes. Extending downwards through the external granular layer, the fibre, on reaching the external molecular layer, becomes considerably attenuated, and gives off a few small horizontal fibres as a contribution to this layer. Proceeding onwards, each fibre in the internal granular layer bears a spherical nucleus. In the internal molecular layer many lateral processes are given off, which largely contribute to the fine granular appearance of this layer. In the ganglionic layer, each fibre splits into two or more limbs, which, passing between the optic-nerve bundles, end in expansions which form a second membrane on the inner surface of the retina, the *internal limiting membrane*.

The structure of the retina at, and in the immediate neighbourhood of, the fovea centralis.—At the centre of the fovea, the rods and the ganglionic layer are absent. The optic-nerve fibres, too, cease to form a definite layer. The bipolar cells of the inner granular layer are diminished in number, and the two limiting membranes are cupped towards each other. Over the rest of the macular region, the cells of the ganglionic layer are considerably increased in number, and are often in six or seven rows. The cones at the margin of the fovea are greatly increased in length and placed very obliquely, their inner ends sloping away from the fovea. On approaching the centre of the depression they become shorter and less oblique, until at the centre itself they are perpendicular and only slightly larger than they are in the rest of the retina.

The structure of the retina at the ora serrata.—Here the layers of the retina terminate almost abruptly, and the innermost coat of the eyeball is continued on as the pars ciliaris retinae. This is composed of two layers of cells—an inner layer of columnar epithelium into which the inner granular layer of the retina passes, and an outer layer of pigmented cells, the continuation of the retinal pigment layer. On approaching the ora serrata, the first structures to disappear are the ganglionic cells and the optic-nerve fibres. These are soon followed by failure of the layer of the rods and cones, and of the outer molecular layer. The fibres of Müller are very large and numerous at the ora serrata.

The *vascular supply* of the retina is derived entirely from the central artery of the retina, with the exception of a slight

anastomosis with the choroidal vessels at the optic disc. There is no anastomosis with the ciliary vessels at the ora serrata. Immediately on leaving the nerve, the artery divides into the superior and inferior papillary branches. Each of these gives twigs to the macular region, the superior and inferior macular arteries, and others which pass horizontally inwards, the median arteries. Frequently, however, the macular and median arteries arise directly from the central artery, and their connection with it is not seen ophthalmoscopically, since it lies behind the optic disc. Each of the papillary branches divides into two, to form the superior and inferior temporal arteries and the superior and inferior nasal vessels, of which the superior temporal branch is the longest, and the inferior nasal branch the shortest. A great number of capillary meshes are formed around the macular and in its outer margin, but no vessels reach the fovea centralis. Each artery has generally a vein accompanying it, so that, as a rule, four chief vessels are seen upon the disc. The superior and inferior papillary veins normally enter the centre of the disc as two separate vessels, their union not being visible with the ophthalmoscope. The larger vessels occupy the nerve-fibre layer, but the capillaries ramify wholly in the middle portion of the retina, and never pass external to the inner granular layer. There are two chief networks of capillaries, one in the inner granular layer, and the other in the layer of ganglion-cells. The outer portions of the retina receive their nourishment from the choroidal vessels, by a process of transudation.

The *lymphatics* of the retina exist around the vessels in the form of perivascular lymph spaces. They can be injected from the optic nerve beneath the pial sheath (Schwalbe).

Physiology of the Retina.—When light falls on the retina, certain changes, mechanical, chemical, and electrical, are produced. The pigment of the pigment epithelium moves forwards to embrace the outer segments of the rods and cones, and, with the pigment, the fine processes of the pigment-cells. At the same time there is a retraction of the cones towards the light. The pigment-cells, from being alkaline, become acid in reaction, and the rhodopsin of the rods is bleached. But besides these mechanical and chemical actions, there are definite electrical changes. Light stimulus is now considered to depend on a series of electro-magnetic disturbances in the all-pervading ether, and when these disturbances, which are rhythmic in occurrence, fall on the retina, a positive electric current passes from the pigment-layer towards the light.

It seems probable that it is in the pigment epithelium that the light stimulus is changed into a nervous sensation, partly by means

of chemical changes, and partly owing to an electrical effect. The impulses produced are then conducted by means of the rods and the cones, through the various layers of the retina, to the nerve-fibres of the optic-nerve fibre layer, and thence by the optic nerve and optic tract to the brain (see fig. 59). Whether the rods and cones have different functions is still *sub judice*; but it seems possible that the former are mainly occupied in conducting nervous impulses produced by white light, while the latter's peculiar use is in the recognition of colours. The effect of these impulses produced in the brain is perceived by the mind as the sensation of light. The organ in the brain with which these are connected being incapable of conveying to the mind any other sensation than that of light, the same sensation is produced whether the stimulation is mechanical, electrical, or what not, and in whatever part of the course of the conducting chain it is applied.

In consequence of the optical properties of the eye and the arrangement of the retinal elements, each of the latter receives light from one point in the visual field, and from no other; this correspondence between the element which is excited and the position of the point from which the light proceeds enables us to judge of the relative position of the points where images are formed in the retina. Our judgment, however, receives some unconscious support from other senses, and many sensations which seem to be simply visual—such as the sense of size, distance, and solidity—are in reality complex, and depend to a certain extent on the teaching of experience, on muscular sense which tells us the position the eyes are in, on the amount of convergence and accommodation used, and on a comparison with well-known objects. The ideas we have of size and distance are very closely related to each other.

For *distinct* vision the image of the object must fall upon the yellow spot, or rather its central part, the fovea centralis. This is called *direct* vision, in opposition to *indirect* vision, which occurs when the image falls on any other portion of the retina. In low degrees of illumination, however, the fovea is less sensitive than the retina in its immediate neighbourhood.

In order that two points may give rise to separate visual impressions their images must be at least 0.003 mm. apart; for, since this is approximately the diameter of the macular cones, images which were nearer together than this would only stimulate one cone, and therefore give rise to but one visual impression.

Over the optic disc there is no retina, and therefore no perception of light—hence this point is called the blind spot, and its existence is shown by the familiar experiment of making a dot and a small

circle about 5 cm. apart, the dot being placed to the left. If, with the left eye shut, the right eye views the dot steadily when held near to and in front of the eye, the circle will usually be also in view. On moving the paper slowly away from the eye the circle will be found to vanish, and on moving it still farther away it will again come into view. When it vanished from sight its image fell wholly on the blind spot. This occurs when the distance of the dot from the eye is about four times that between the dot and the circle. The percipient elements of the retina are the rods and cones, especially their outer segments. This is proved by the facts, first, that only cones are found over the fovea centralis; secondly, that the vessels of the retina can be perceived entoptically under certain conditions. If a thin metal disc, having a pinhole aperture at its centre and a piece of pale blue glass behind it, be rapidly moved in a small circle in front of the pupil while we steadily look through the pinhole at a white cloud, a complete outline of all the network of capillaries around the fovea centralis can be speedily obtained. Purkinje's experiments, as described in textbooks of physiology, also show the existence of the blood-vessels to be in front of the sensitive elements of the retina.

Corresponding retinal areas.—In order that the two retinal images of an object may give rise to a single visual impression, it is necessary that images should fall upon corresponding retinal areas. Thus the upper halves of the retinæ correspond, and also the lower halves; but the nasal side of one retina corresponds to the temporal side of the other, and *vice versa*. When we see (in indirect vision) to the left side, it is not so much with the external part of the right as with the internal part of the left retina, and *vice versa*. Now all rays affecting the external aspect of the retina come from the nasal *visual field*; and this field, tested separately for each eye, is always found deficient in extent compared with the temporal visual field, even when the influence of the projecting bridge of the nose is eliminated (Landolt). It is probable that it is only when the images fall near the central part of the retina that they continue to form a single visual impression; when one falls near the centre, and the other on a very peripheral part, the latter image, being less intense, is disregarded.

The Ophthalmoscopic Appearance of the Retina.—When the healthy fundus is examined by the direct method, and with a bright illumination, the retina is, in the majority of cases, found to be perfectly transparent. It reflects little or no light and offers no resistance to that reflected from the choroid, and is therefore quite invisible—in fact, were it not for the presence of its blood-vessels, it

would be impossible by this test alone to assert that the retina existed. When only a feeble illumination is used, a slight brilliant reflex can be obtained from the region immediately surrounding the optic disc. This is caused by reflection of the light from the curved surface, where the fibres of the nerve are spreading out to reach the retina. It is seen if the nerve-fibre layer is thicker than usual, and especially where the fibres cross a vessel. The appearance is difficult to describe: it varies with each tilting of the mirror, is somewhat like the reflection from shot-silk, and is lost in the red reflex from the choroid when the intensity of the light is increased. In certain cases, however, there is a retinal reflex of this nature whatever the intensity of the illumination; this usually obtains in young children, and in persons of very dark complexion, where the choroid is highly pigmented. Along the course of the vessels also this may be usually observed in the form of a bright line; it is distinguished from a pathological change by the fact that the reflection will shift from one side of the vessel to the other by the slightest movement of the mirror. Other reflex phenomena are occasionally seen. *Gunn's* or *Crick's dots* consist of a few bright dots arranged in groups in the macular region, and best seen in the eyes of dark-complexioned children. *Metallic dots* differ from the above in not being arranged in groups, and in not being confined to the macular region. All these reflex phenomena are probably due to slight irregularities on the surface of the retina.

The *yellow spot* is to be sought for on the outer side of the optic disc, at a distance equal to twice the diameter of the latter. In many cases, especially in adult fair persons, the healthy so-called yellow spot presents the appearance shown in fig. 1, opposite p. 198; that is, it differs but little, if at all, in colour from the surrounding fundus, and can only be distinguished from the other parts of the retina by the absence of visible vessels, and by its position with regard to the optic disc. In the majority of eyes, however, there is an intensification of the colour, giving an appearance similar to that shown in fig. 2, opposite p. 198. In some cases, more especially in young subjects, and dark eyes, a small, yellowish, somewhat brilliant spot is seen; this is the fovea centralis. It is surrounded by an ill-defined dark area, and around this again there is sometimes a greyish halo, which changes its appearance when the mirror is tilted.

The vessels of the retina are easily distinguished from those of the choroid by their radiating course, their dichotomous mode of branching, their clearness of tint, and their well-defined outline.

The peripheral as well as the central portions of the retina should always, as far as possible, be examined; these are better seen when

the pupil has been previously dilated. The ophthalmoscope should be held as close to the cornea as possible (about 5 mm.), and the patient told to look successively in the outward, inward, upward, and downward directions; this brings into view the outer, inner, upper, and lower portions of the retina respectively. In order to see the lower portion when the patient looks downwards, it is necessary to elevate the upper lid with a finger of the hand which is not holding the ophthalmoscope. The examination of the peripheral parts of the fundus is especially important, because certain morbid conditions—such as pigmentary retinitis, disseminated choroiditis, detachment of retina, and other affections, often make their first appearance in that part, while the central portions are apparently normal.

The arteries of the retina are from two-thirds to three-fourths the size of the veins, they are lighter in colour, their course is somewhat straighter, and they are recognised by the presence of a fine bright line on each side of the vessel.

Pulsation of the retinal veins is sometimes observed, even in normal eyes. This is only seen upon the optic disc, and various theories have been propounded as to its causation.

1. Donders considered it to be owing to the rhythmically increased arterial tension communicated to the veins, the vitreous body being less compressible than these. It occurs only on the disc because here the tension of the veins is most feeble. The pulsation thus appears in the diastolic arterial interval.

2. Schön ascribes it to the pulsation of the artery communicated to the vein as they lie together in the optic nerve.

Pulsation of the retinal arteries is very rarely found in normal eyes, although both arterial and venous pulsation can be produced by digital pressure upon the globe during ophthalmoscopic examination. When it does exist it may be due to one of two local causes: (i) It may be nothing more than a pulsation communicated from the neighbouring vein, in which case the pulsation of the artery would succeed that of the vein. (ii) It may arise from the fact that the branches at the disc are given off at right angles to the main trunk immediately after its bifurcation (Otto Becker).¹ With these rare exceptions, therefore, the existence of arterial pulsation at the optic disc is indicative of some pathological condition either of the eye itself, of the orbit, or of the general circulation—*e.g.*:

1. Arterial pulsation may be caused by increased intra-ocular pressure which prevents the retinal arteries from becoming filled except at the acme of the pulse-wave. It is not uncommon in

¹ *Archiv für Ophth.* vol. xviii. part i. p. 266.

glaucoma; and when not occurring spontaneously in glaucoma, it can usually be temporarily produced by very slight pressure upon the globe, whereby the tension is still more increased.

2. It is also occasionally seen in cases where the trunk of the central artery has become compressed, as in certain cases of optic neuritis, and in tumours of the orbit where the optic nerve is pressed upon, and of the optic nerve itself.

3. The tension of eye being normal, arterial pulsation may be caused by low arterial tension, arising from deficient action of the heart. It is common in aortic regurgitation with hypertrophy of the left ventricle, in Graves's disease, in syncope following loss of blood, in general anæmia, and it is said to be present during the period of asphyxia in cholera.

CONGENITAL ANOMALIES.

Many changes are seen in the fundus oculi which exist at birth, and persist throughout life without changing in appearance or producing any deterioration in visual acuity or visual range. Such anomalies may be mistaken for diseased conditions, and consequently their recognition is essential in order to be able to arrive at a correct diagnosis. They may be present in the vascular coat—the *choroid*; or in the nervous tunic—the *optic disc and retina*. It is well to bear in mind that the retina, disregarding its pigmentary epithelium, is a transparent membrane with the exception of the blood contained within its vessels.

Congenital affections of the choroid have already been described, including changes in its colour and appearance, coloboma, and congenital crescent (p. 227). Though coloboma is described under Choroid, it must be remembered that the primary cause of the deficiency is a non-closure of the choroidal fissure, a cleft in the lower part of the optic cup, an epiblastic formation; this developmental deficiency in the nervous structure is attended with a similar flaw in the development of the choroidal coat. A coloboma of the choroid is, as a rule, associated with a *coloboma of the retina*, and a loss of a corresponding area of the upper half of the visual field. The retinal vessels ramify over the exposed sclerotic coat, usually supported in an indefinite connective-tissue membrane (see fig. 2, opposite p. 227).

The following include most of the congenital peculiarities of the optic disc and retina.

Coloboma of the Optic Disc.—This may be present with or without coloboma of the choroid. A large deep depression is seen involving a larger or smaller portion of the disc. It is due to the non-closure of the fœtal cleft in the optic nerve.

Opaque Nerve-fibres.—As we have seen (p. 242), the normal optic-nerve fibres at the posterior part of the lamina cribrosa become, as a rule, entirely deprived of their medullary sheath, and are quite transparent both in the papilla and in the retina. In some cases, however, it is found that the medullary sheaths are regained as the optic fibres leave the disc, and may be seen with the ophthalmoscope as opaque brilliant white patches, occupying more or less of the area and circumference of the disc, and extending towards the periphery of the fundus in cometlike processes. Occasionally the whole disc is covered by opaque nerve-fibres; in these cases the medullary sheaths are probably never lost. Sometimes only a single patch exists, forming a snowy-white spot on the edge of the disc; in other cases there are several of these; more usually, however, the opaque nerve-fibres are most visible where the fibres are naturally most abundant—that is, in the directions of the chief divisions of the retinal artery. A clear space has been seen in a few cases to intervene between the patch and the disc, and, rarely, the patch has been observed to occupy a position at a considerable distance from the papilla. In many cases the fibres have a distinctly fibrillated appearance, more especially towards their free edges. They can be distinguished from morbid products by their brushlike extremities and the fact that *they are in front of the retinal vessels*, and that some part of the retinal artery can be seen to be embedded, as it were, in the midst of the opaque fibres (see fig. 2 on opposite page). They hardly ever occupy the region of the yellow spot. Cases have been recorded in which, with the onset of optic atrophy, patches of medullated nerve-fibres have disappeared. Microscopical examination of a few cases of opaque nerve-fibres has shown that varicosities on the fibres exist in the patches. These varicosities are either thickenings

Plate VII.

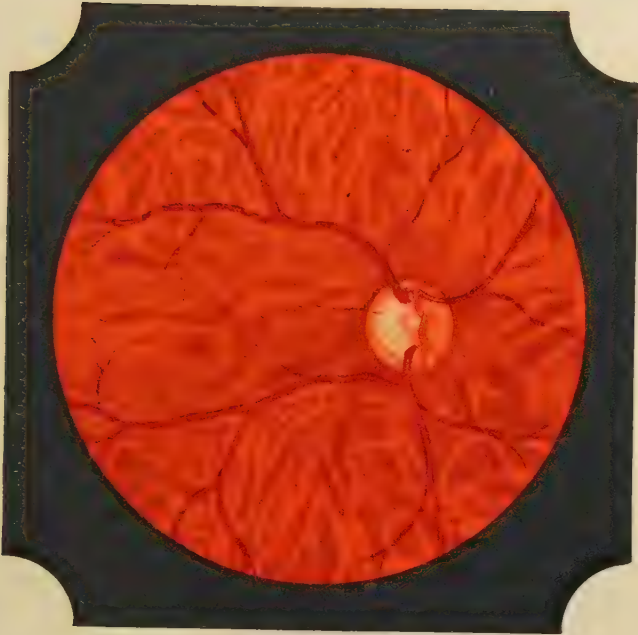


Fig. 1.—Physiological cupping of Optic Disc.

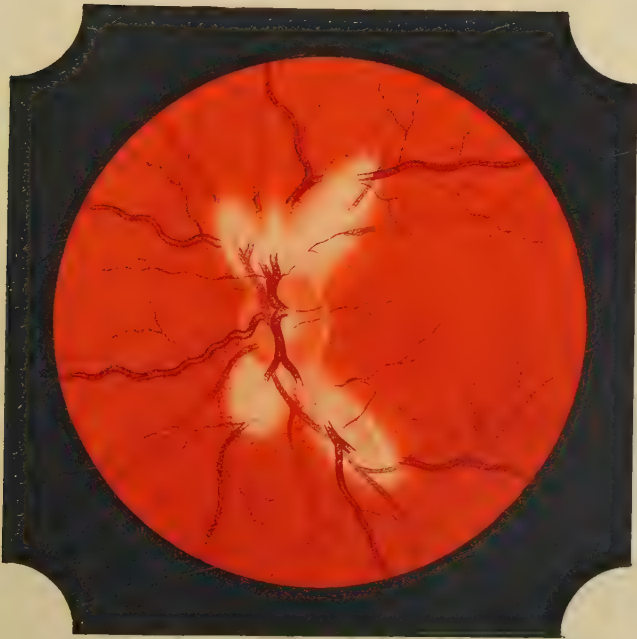
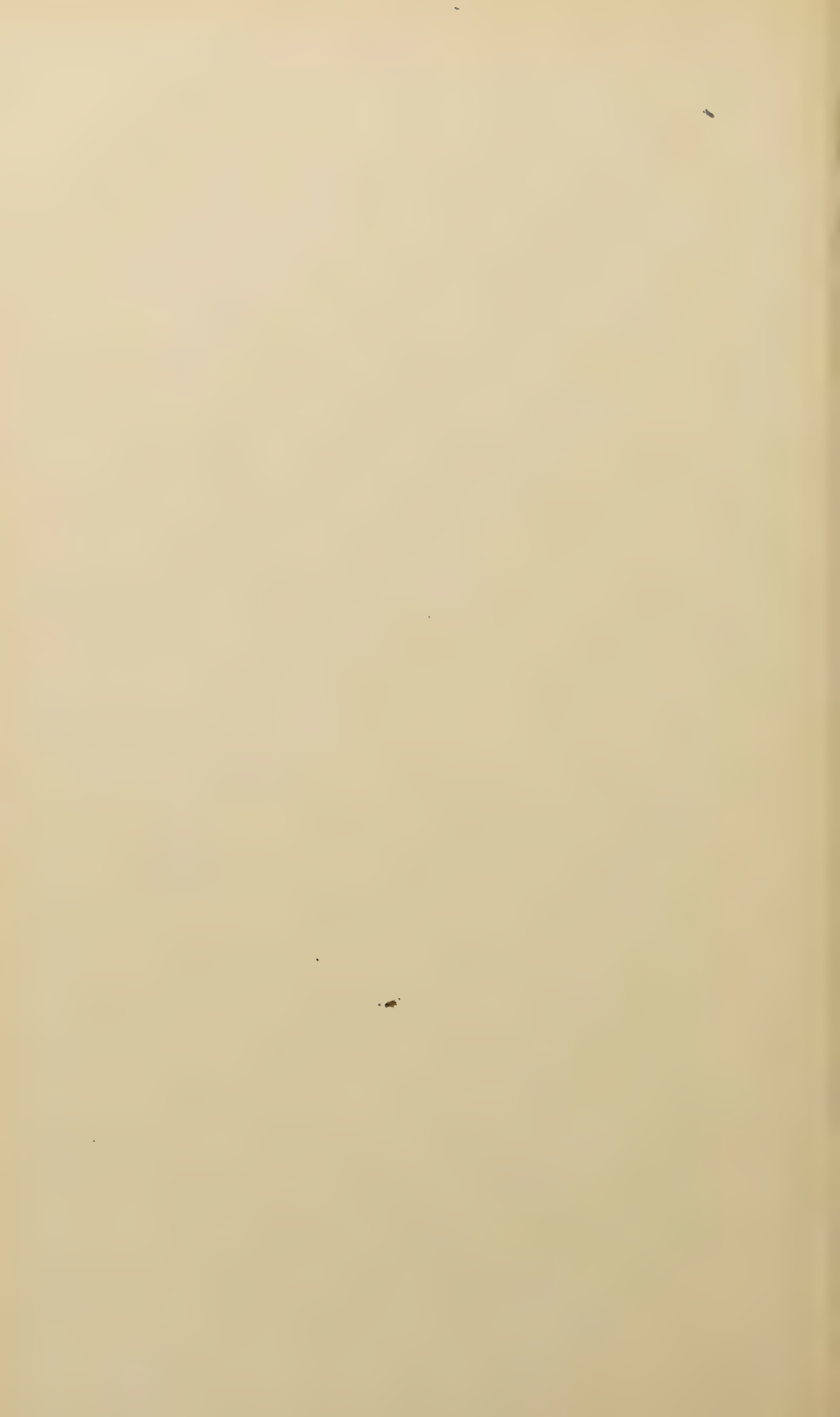


Fig. 2.—Opaque nerve fibres.



on the axis cylinders, or of the medullary sheaths, or perhaps they are collections of fluid between the two.

Opaque nerve-fibres are apparently often associated with imperfect mental development.

Visual acuity may be quite normal as far as the opaque fibres are concerned, although this affection is often accompanied by other abnormal conditions, as hypermetropia, astigmatism, &c., which may cause deterioration of vision. The *visual field* also is normal, with the exception of the blind spot, which is generally large and irregular in proportion to the extent of the patch or patches of opacity.

Vascular Peculiarities.—(a) The artery may emerge as the two papillary branches. (b) Bifurcation of retinal veins is a rare condition occasionally seen; the division usually occurs near or on the disc. (c) The retinal artery, instead of being on the nasal side of the vein, may be on its temporal side. (d) Arteries and veins may be twisted round each other to form one or more loops, a condition especially found in hypermetropia. (e) Cilio-retinal vessels are sometimes present, and are recognised by appearing or disappearing at the margin of the disc, and by not being connected with the central vessels. If they be traced towards the disc, they will be seen to bend away peripherally. They are usually small, and are not as a rule attended with much diminution in size of the *arteria centralis retinae*. It is often difficult, however, to be certain that they are not branches of the central vessel arising within the optic nerve. In rare cases the central artery is very small, and almost entirely replaced by cilio-retinal vessels. They are derived from the short posterior ciliary arteries. (f) White lines along the vessels may exist, and be mistaken for pathological changes, such as are seen after papillitis or neuro-retinitis. They are due to a prolongation of the connective tissue which supports the vessels while in the substance of the optic nerve beyond the lamina cribrosa. This tissue does not extend beyond the margin of the disc; in post-papillitic changes the white lines are invariably seen beyond the edge of the disc. (g) Remains of the hyaloid artery. The capsulo-pupillary membrane, which surrounds the foetal lens to nourish it, is vascular and receives its blood-

supply from a branch of the arteria centralis retinæ, the hyaloid artery, which traverses the vitreous in a special canal (canal of Stilling or Cloquet) to the posterior surface of the lens, where it breaks up into numerous branches, and supplies the membrane. Sometimes this vessel does not undergo complete atrophy. A few fibrous filaments may be seen floating in the vitreous, being attached posteriorly to the centre of the disc. Occasionally, the remains will extend to the posterior surface of the lens, at the posterior pole of which a white patch (posterior polar cataract) will be seen. Small masses of connective tissue are, rarely, seen on the disc; these are probably remnants of the hyaloid artery. Sometimes a small portion of the vessel will remain pervious with circulation through it; in such a case it appears in the form of one or more vascular loops. Newly formed vessels in the vitreous may be mistaken for persistent remains of the hyaloid artery; they differ, however, in being numerous and, as a rule, associated with some gross fundus lesion. (*h*) The vessels may be unusually tortuous. This is often found accompanying some blurring of the disc margin and a high degree of hypermetropia. It is probably due to the early retinal folds persisting, and resembles in some respects an early papillitis, from which it must be carefully distinguished. (*i*) Various other vascularities are sometimes seen. Thus, one or more of the arteries may be twisted round its corresponding vein. Loops may be seen leaving the disc either laterally or antero-posteriorly, and almost immediately returning. Branches of anastomosis between an artery and its vein may be present.

Pigmentary Deposits.—Marginal pigment at the disc has already been referred to; it is more usual to see it at the outer side, though it may completely encircle the disc—*choroidal ring*. Pigment on the disc is rarely seen. Peripheral pigmentation is a congenital anomaly which is sometimes met with, and is unattended with any visual defect or alteration in the disc or retinal vessels. Some interesting cases of this condition have been recorded by Sydney Stephenson¹ and others. The deposit involves a sector of the fundus, and is in the form either of dots or of larger plaques

¹ *Trans. Ophth. Soc.* vol. xi. p. 77.

(so-called *moles*). It is non-progressive, asymmetrical, and not attended with symptoms or other signs suggestive of pigmentary degeneration of the retina.

The Physiological Cup.—On examination by the direct method, the optic disc frequently presents an excavation at its centre, just at the point of emergence of the central vessels. This is due to exposure of the central part of the lamina cribrosa by the divergence of the nerve-fibres (see fig. 1, opposite p. 254). The central hollow thus formed is known as the physiological cup or pit; it is usually funnel-shaped, and its nasal side is steep, while it gradually slopes up on its temporal side. The lamina cribrosa, with a sievelike mottled appearance, may be seen at its bottom. It varies considerably in extent and depth, but it never extends to the extreme edge of the disc, as is the case in glaucomatous cupping. Parallaxic movements can often be obtained. It does not interfere with visual acuity. The vessels usually curve round the margins of the cup, ascending along its sides; they may, however, arise from the bottom and pass straight forwards. More rarely they pierce the sides, and still more rarely arise from the uncupped part of the disc.

The scleral ring is another feature of the normal disc which is frequently observed. It is a whitish ring, situated at the edge of the disc, which is caused by the aperture in the choroid being somewhat larger than that in the sclerotic, so that the edge of the latter is seen as a white band through the transparent nerve-fibres. It is often more visible on the outer side of the disc than throughout the rest of its extent, owing to the fact that the optic-nerve fibres are thinner at that part. It can generally be seen as a complete ring when the optic-nerve fibres are atrophied, as in chronic glaucoma and grey atrophy of the disc.

The optic disc is usually circular in form; in some cases, however, it appears somewhat elongated in one direction. This may be its real condition, in which case its form will be the same in whatever way it is examined—namely, with the longest diameter vertical; or the oval appearance may be due to astigmatism. If due to astigmatism, examination by the indirect method will show that the direction of its long axis and its

length compared with the short axis vary with the position of the lens, while if examined by the direct method, its long axis will be at right angles to the direction as appears by the indirect method. The average diameter of the disc is about 1.6 mm. ; its apparent size varies with the refractive condition of the eye. (See Refraction.)

HYPERÆMIA OF THE OPTIC DISC.

Hyperæmia of the disc is characterised by increased redness. The large central vessels can be plainly seen, but the colour of the area of the disc is intensified ; in severe cases its redness is only with difficulty distinguished from that of the surrounding region (see fig. 1, opposite p. 262). On the other hand, the physiological cup, if present, is very obvious. It is, as a rule, unattended by impairment of the visual function, although there may be hypersensitiveness to light (photophobia), early fatigue in reading, and indistinct pain in and around the globe.

Hyperæmia is frequently found in the subjects of hypermetropia and hypermetropic astigmatism, and is liable to be mistaken for actual papillitis. It is also common amongst those who are obliged to work for long periods in a bright light, such as gaslight. It is usually seen in the early stage of optic neuritis. It is found in some cases of sympathetic ophthalmitis in the sympathising eye, and is due to congestion of the vessels derived from the short ciliaries which also supply the choroid.

In some cases the congestion is of a passive or venous nature, resulting from obstruction to the return of blood to the heart ; under these circumstances the veins of the disc are tortuous and distended, and its colour is deeper than that which occurs in active hyperæmia. A very definite form is hysterical hyperæmia, found especially in girls at puberty.

In the *treatment* the cause of this affection must be borne in mind. Any existing error of refraction must be corrected by suitable glasses. Over-use of the eyes must be discontinued, and bright light avoided by the use of neutral-tinted glasses.

OPTIC NEURITIS.

Optic neuritis is divided into two great classes: (a) disease of the retro-bulbar portion of the optic nerve—*retro-bulbar* or *retro-ocular neuritis*; and (b) disease of the nerve-head or papilla—*papillitis*.

Retro-bulbar Neuritis.—*Etiology and pathology.*—The whole subject of retro-bulbar neuritis is one of extreme difficulty, and much confusion still exists as to what cases should be included under the term. It is still the custom to exclude the so-called toxic amblyopias, although a retro-bulbar neuritis is perhaps the pathological cause of the condition. These amblyopias differ in their symptoms from other forms of retro-bulbar neuritis, and are consequently grouped together and will be described under a separate heading. Many neurologists consider the group of symptoms found in retro-bulbar neuritis to be indicative of incipient insular (disseminated) sclerosis. Ophthalmologists, on the whole, differ from this view, though there is no definite consensus of opinion as to what are the chief etiological factors. Some of the causes to which retro-bulbar neuritis has been attributed are the following:

1. Orbital cellulitis. This may have its origin in inflamed sphenoidal or ethmoidal cells, the result of a catarrh or an injury; it may possibly arise from carious teeth.

2. Orbital periostitis the result of a chronic syphilitic inflammatory process, especially in the neighbourhood of the apex of the orbit, and spreading to the optic nerve and its sheaths.

3. A syphilitic gumma of the optic nerve itself.

4. Tuberculous disease of the optic nerve and chiasma.

5. Gout and rheumatism.

6. Insular sclerosis. It is said to be found in 50 per cent. of cases of this disease.

7. Migraine.

8. High arterial tension.

9. Toxines. See Toxic Amblyopias (p. 328).

The site of the lesion in retro-bulbar neuritis has been definitely proved to be the papillo-macular group of fibres of

the optic nerve, which pass between the outer part of the disc and the macula. There is, however, a difference of opinion as to whether the disease is an interstitial inflammation or a primary degeneration. The pallor of the disc is probably not due to a true atrophy of the nerve-fibres, since vision is often good with a very pale disc. It may be due to some constriction of the blood-vessels by the contracting fibrous tissue formed as a result of the inflammatory process, or it may be the direct result of a hyperplasia of the normal connective tissue of the optic-nerve head.

Symptoms.—Retro-bulbar neuritis, in its typical form, has a very definite symptomatology, though these symptoms vary very considerably in their intensity. Atypical forms, too, are frequently met with. The onset of the disease is often sudden, the patient finding a defect in his vision on waking up from sleep. In almost all cases the disease is confined to one eye, though the other eye may be subsequently affected.

Loss of sight. It is for this that the ophthalmic surgeon is consulted, and he may find that the eye is absolutely blind, even perception of light being lost. On the other hand, a vision of 6/6 Snellen may be obtained, though with great difficulty, the patient seeing each letter as if through a thin veil. Between these two degrees all grades are found. The early stages of the disease are often characterised by transient loss of sight, with complete or almost complete recovery in the intervals.

Pain. This is frequently felt during movements of the eyeball, and especially in the upward direction. The globe may be tender to backward pressure.

Altered pupillary reflex. In all cases, except the very slight ones, the direct pupillary reflex to light is affected. When the sight is only partially lost, this reflex is present, though the contraction of the pupil to light is not sustained, oscillatory movements being seen; and the pupil comes to rest in a condition of dilatation, though the stimulus is not removed. In the severe cases, however, where all perception of light is lost, the direct pupillary light reflex is completely lost, but in all cases the consensual light reflex is present.

Alterations in the visual fields. The commonest condition

is an absolute central scotoma, varying from 10° to 20° in diameter. This is often of the positive variety, the patient complaining of a mist before objects looked at. The visual fields for white and colours are, as a rule, not peripherally contracted. There may, however, be many varieties in the way the visual fields are affected. Peripheral contraction may take the place of, or may accompany, a central scotoma. Again, reds and greens may be completely lost, with an absolute central scotoma, and no peripheral contraction of the field for white and blue. Occasionally the scotoma is eccentric; and rarely it is only relative, white and blue being visible all over the field.

Ophthalmoscopic signs. These are often absent in the earlier stages of the disease. Sometimes, however, after comparison with the affected eye, a slight blurring of the disc outline is seen, which in a few cases is well marked, the whole disc having a hyperæmic appearance. After the disease has been present for some time, the outer part of the disc becomes somewhat pale. This is a very characteristic and constant sign, and in almost all cases persists. It is often difficult to distinguish this from the normal disc with a well-marked physiological cup; but a careful comparison between the two eyes must be made, and in this way a definite diagnosis between the two conditions is usually possible. Later, this pallor may spread until the whole disc is involved; this, however, is rare.

Diagnosis.—The diagnosis of retro-bulbar neuritis is often of extreme difficulty, on account of its very few objective signs. The behaviour of the pupil to light is of extreme importance, since any impairment of the reflex excludes functional forms of amblyopia. Again, the form of the visual field is of great assistance. Hysterical amblyopia has a peripherally greatly contracted field with no scotoma, quite unlike the typical uncontracted field of retro-bulbar neuritis, with its central scotoma. Toxic amblyopia is almost always binocular, and the central scotoma is very rarely absolute. Incipient primary optic atrophy has a slightly contracted field, especially for colours, with no scotoma. Again the ophthalmoscope must be very carefully used to exclude all fine macular changes,

whether retinal or choroidal, which are liable to be passed over in a hurried examination of the fundus. Any pallor of the disc points to organic, and not functional, disease. Should papillitis be present, intracranial disease must be excluded. A careful study of the history will usually suffice. In retro-bulbar neuritis the vision fails early and centrally, improving rapidly with subsidence of the papillitis, leaving some pallor of the temporal portion of the disc. In papillitis due to intracranial disease, however, the vision may be little or not at all affected until the papillitis is well marked; subsequently, the visual acuity becomes greatly diminished, with total white atrophy of the disc.

Prognosis.—The prognosis of the disease is often good, and especially is this the case where there has been a rapid onset and a rapid development. A complete cure may take place, though it is common to find some slight permanent central scotoma. Permanent blindness is never the result; instances of a partial recovery have been recorded where perception of light has been totally lost for three or four weeks.

Treatment.—Iodide of potassium and mercury should be administered in some form, together with the application of blisters to the temple. Later, nerve-tonics are indicated. Santonin has been considered to be a retinal stimulant, and is consequently used by some. The eyes should be kept at rest and shaded from bright light, and the habits of the patient carefully regulated.

Papillitis.—Following the suggestion of Leber, we propose to use the term ‘papillitis’ in preference to ‘choked disc,’ ‘descending neuritis,’ and other terms, which are misleading as involving theories of causation not yet proved.

The ophthalmoscopic signs of papillitis.—During the active stage the appearances are quite characteristic, and are as follows:

1. *Loss of the outline of the disc.*—The margin of the disc, which in a healthy fundus is so well defined, beginning at the nasal side, becomes gradually indistinct until it completely fades out of recognition. Congestion alone will cause the disc-outline to become obscure by changing the paler colour of the papilla to a hue similar to that of the surrounding fundus.

Plate VIII.

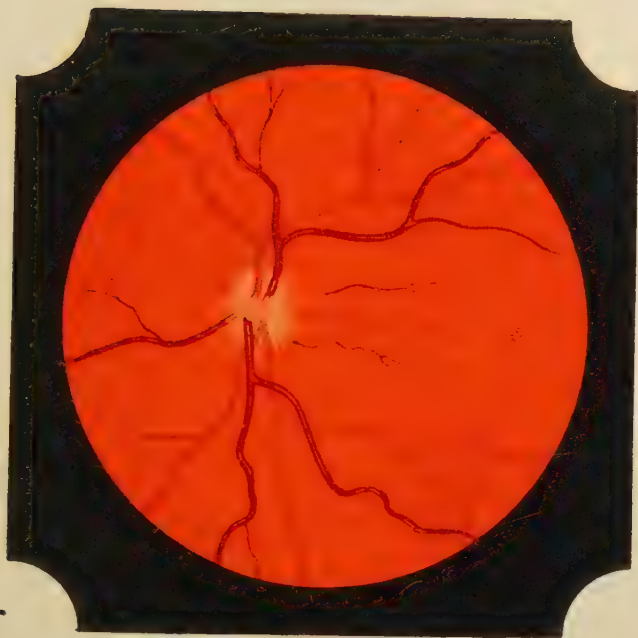


Fig. 1.—Commencing Optic Neuritis.

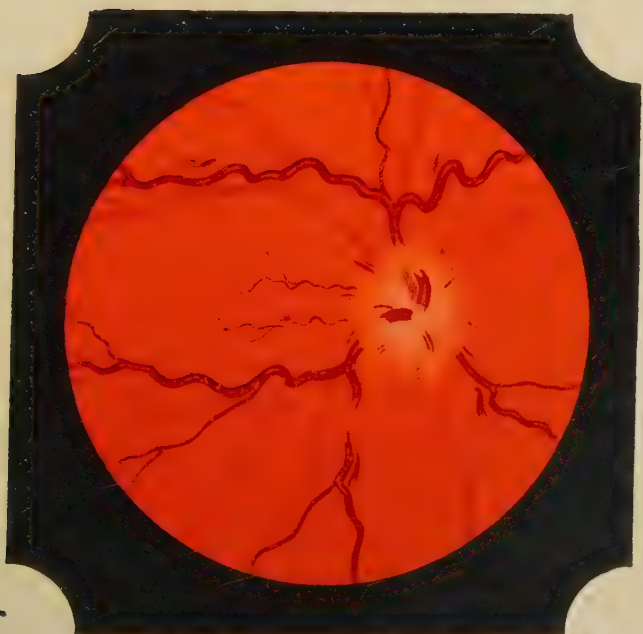


Fig. 2.—Neuro-retinitis with hæmorrhages.



When the second stage of inflammation sets in, the exudation entirely obliterates any existing trace of the disc. Its position is recognised by the entry and exit of the retinal vessels, also by a characteristic radial appearance which is often seen at the site of the hidden disc. These radii are caused by the fibres of the optic nerve, which spread out to form the nerve-fibre layer of the retina. In health they are transparent and invisible; inflammation renders them swollen and semi-opaque from the imbibition of inflammatory œdema.

2. *Change in colour.*—It has already been mentioned that the disc becomes redder than natural from hyperæmia; this redness, with the radial streaks, remains throughout the acute attack. Subsidence of the inflammation is shown by the centre of the papilla becoming pale, and eventually white; this spreads peripherally, until a pale woolly white disc remains with an indefinite margin (consecutive atrophy).

3. *Swelling of the disc.*—Since the radial appearance around the entry of the arteria centralis retinæ extends outwards beyond the margin of the normal disc, it is customary to say that the disc is larger than natural. Again, if the direct method of ophthalmoscopic examination is adopted, it will be noticed that the details at the papilla are more clearly made out by the use of convex lenses; with the same lenses, however, with which the disc can be seen the details of the fundus of the periphery are blurred and out of focus, and the convex lenses must be reduced before the peripheral vessels are clearly seen. The cause of this is an undue prominence of the papilla, or nerve-head, from œdema and cellular infiltration. So great is the swelling of the disc in some cases that it can be seen distinctly with a lens as high as +6D. The adjacent optic nerve, with the swollen disc, has been aptly compared to a champagne-cork.

4. *Interruptions in the course of the vessels.*—The exact point of entry of the retinal vessels is often obscured, and the arteries may be so hidden from view by the inflammatory exudation as not to be seen at all in the region of the disc, only appearing at the periphery of the striations. The veins, however, can usually be seen following a serpentine course through the œdematous papilla, here and there dipping down

into the exudation which temporarily hides them from view. The exudation often produces a general haziness of all the details in the neighbourhood of the disc.

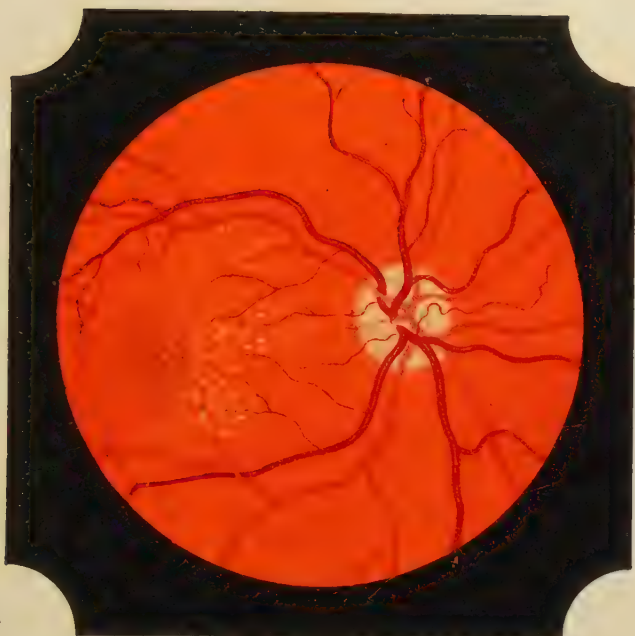
5. *Distension and tortuosity of the retinal veins.*—The exudation at the papilla retards the venous return and impedes to a less degree the arterial inflow. Mechanical congestion therefore occurs, and the veins consequently become distended and tortuous, and as they approach the swollen nerve-head they are seen to bulge forward and bend abruptly as they course to the centre of the papilla. The arteries, beyond being lost in the region of the disc, do not present any marked peculiarity; in severe cases they may be smaller than normal.

6. *Hæmorrhages.*—In severe papillitis, hæmorrhages are not uncommonly seen in the neighbourhood of or upon the disc, usually along the course of the vessels, and at the periphery of the striations in the form of small dots, or linear 'torch-like' extravasations; large hæmorrhages are rare. Soft woolly white patches may be seen on or near the disc: these are old hæmorrhages, in which the colouring matter has been absorbed, whilst the coagulum crowded with leucocytes remains; they eventually become absorbed.

This affection is seldom confined to the papilla; it can usually be observed to invade more or less of the surrounding retina. Macular retinitis is very often found in papillitis the result of intracranial, and especially cerebellar, tumour. It is very similar in appearance to albuminuric retinitis.

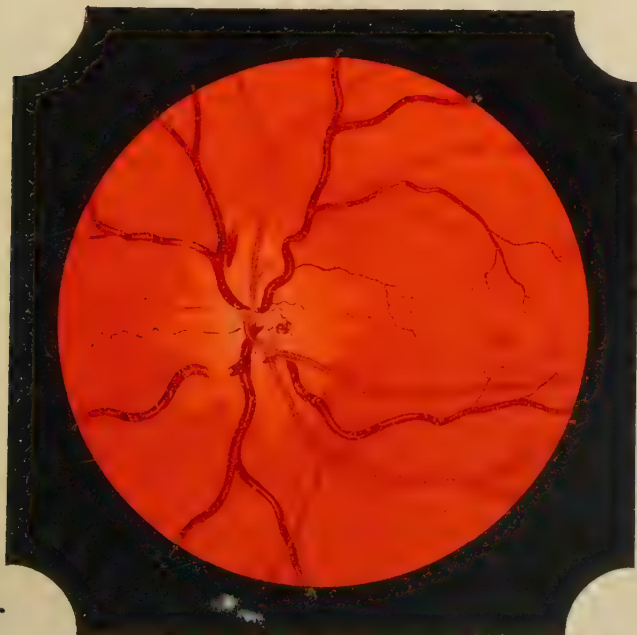
In the post-papillitic stage these changes in the appearance of the disc undergo gradual subsidence. At the end of some weeks or months the opacity begins to disappear, and the edge of the disc may be seen as through a mist ('woolly disc') which gradually becomes less and less. The edge of the disc is thus again brought into view, and may present the same appearance as it did before the papillitis, or its outline may be somewhat irregular. The vessels gradually become less tortuous, and may undergo permanent contraction. White spots arranged in lines are sometimes seen in this stage radiating from the yellow spot. They appear only after severe papillitis; they differ from those of albuminuric retinitis in that they occur only to the inner side of the macula lutea, not, as in the latter affec-

Plate IX.



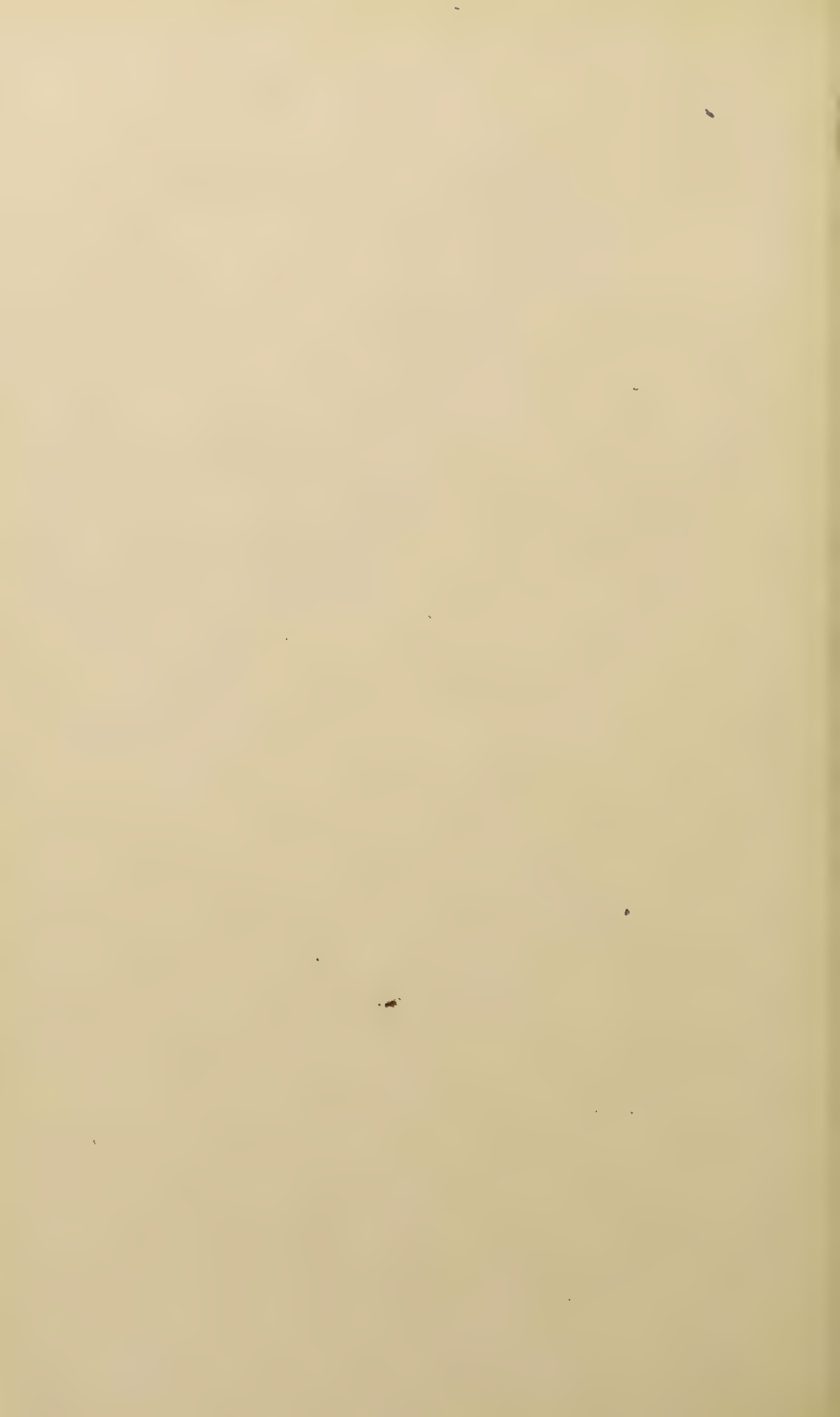
A.W.H.

Fig. 1.—Macular changes with Atrophy.



W.J.R.

Fig. 2.—Optic Neuritis.



tion, all round it; and they tend to disappear spontaneously after the lapse of a few months (see fig. 1, opposite p. 264). The area of the disc may resume its normal pinkish tint, or may be more or less blanched. (See Atrophy.)

Other symptoms of papillitis.—It is remarkable that considerable swelling and haze of the optic disc may exist before the patient experiences any serious interference with vision. Thus, there may be unimpaired visual acuity, good colour-perception, and an unrestricted visual field.

Vision is seldom much impaired until papillitis has existed for some time. If resolution take place quickly—that is, before the inflammation has given rise to atrophy of the nerve-fibres—there may be no failure of vision at all; or the sight, having become affected even to a serious degree, may quite recover. As a rule, however, it is common to find papillitis attended with considerable derangement of vision: (1) *Visual acuity* may be much impaired, or may even be reduced to mere perception of light. (2) *Colour vision*, more especially for green and red, may be considerably interfered with. (3) The *visual field* may be found to differ from the normal in various ways. The blind spot—that is, the scotoma corresponding to the optic disc—is usually enlarged. The field for white may be but slightly, if at all, contracted; whilst the field for green may be much diminished or entirely lost. The field for red may also be diminished. These changes in the visual field become more marked as the atrophic changes set in.

Both eyes are usually affected, but the vision is generally worse in one eye than in the other at the same time. The appearance of a central scotoma for colours (such as is found in tobacco amblyopia) is rare. Failure of vision usually comes on gradually; in some cases, however, it has been known to be very considerable in the course of a few days.

When blindness supervenes, as is unfortunately often the case, it generally does so gradually.

Pathology of papillitis.—If we examine the optic nerve microscopically, by making horizontal and vertical sections through the region of the optic disc of an eye removed during the acute stage of papillitis, we find all the trabecular tissue, the neuroglia, and the blood-vessels, infiltrated with freely

staining nuclei. There is often, also, considerable œdema of the trabecular tissue. The intersheath space of the optic nerve is also affected; it may be distended with fluid, and contain only a few inflammatory nuclei, or there may be little or no fluid, but many nuclei. The intraocular portion of the nerve (papilla) is found to be swollen, and to bulge forwards into the vitreous cavity. In thus starting forwards, it often causes separation of the retina from the choroid near the edge of the disc, so as to give to the section of the inflamed papilla a pedunculated appearance.

This condition of hypernucleation of the nerve, nerve-sheath, and papilla is more marked in cases of meningitis than in papillitis arising from other causes. Observations,¹ however, tend to prove that in all cases of papillitis there can usually be found more or less hypernucleation in the optic nerve-trunk, as well as in the papilla; this may be more abundant at the disc than in the nerve-trunk, or *vice versa*.

The causes of papillitis.—(1) *Intracranial diseases* are by far the most frequent; they are said to give rise to at least four-fifths of the cases of papillitis (Mauthner). Of these the most common is cerebral tumour, though it is very usually found in meningitis. It occurs in certain epidemics of cerebro-spinal meningitis. Then follow cerebellar tumour, abscess of the brain, hydatid-disease of the brain, cerebral softening from vascular obstruction, intracranial aneurysm, that of the middle meningeal artery being the most common, and thrombosis of the cavernous sinus.

As to the variety of cerebral or cerebellar tumour, no one form seems to be especially liable to set up papillitis. It is common in glioma, glio-sarcoma, gumma, and in tuberculous granuloma. In almost every case of papillitis due to intracranial disease the condition is bilateral, though it commonly begins somewhat earlier in one eye. No help in the localisation of the tumour can be got from a knowledge as to which eye failed in visual acuity first. A cerebellar tumour often causes, in addition to the papillitis, a stellate appearance at

¹ Vide Gowers on *Medical Ophthalmoscopy*, 1882; also Brailey, Walter Edmunds, Stephen Mackenzie, and Leber in the *Trans. Internat. Med. Cong.* 1881; and the *Trans. of Ophth. Soc.* 1881 and 1882.

the macula very similar to that found in albuminuric retinitis.

(2) *Intraorbital diseases*.—The chief of these are cellulitis, periostitis (usually syphilitic in origin), tumours of the optic nerve (see p. 302), thrombosis of the central vein, and intravaginal hæmorrhage. Papillitis due to any of these causes is unilateral.

(3) *General affections*.—Of these the most important are chronic Bright's disease, uræmia, glycosuria, the various forms of anæmia, the acute specific fevers, especially typhus fever and influenza, syphilis, and lead poisoning. As rare causes may be mentioned menstrual irregularities, acromegaly, epidemic cerebro-spinal meningitis, disseminated sclerosis, acute spinal myelitis, tetanus, multiple neuritis, and poisoning by iodoform and alcohol. An interesting though rare cause of papillitis is a severe hæmorrhage, such as hæmatemesis, post-partum hæmorrhage, and epistaxis. A thrombus in the central artery of the retina is probably the usual cause.

The theories as to the cause of papillitis in cerebral disease are chiefly as follows :

1. The mechanical theory of von Graefe assumed venous obstruction from increased intracranial pressure affecting the cavernous sinus. This view is now abandoned, because free anastomosis has been demonstrated between the orbital and facial veins, and because large tumours of the brain may exist with very little papillitis ; besides, thrombosis of the cavernous sinus is not always attended with papillitis, which should be the case if this theory was correct ; while, on the other hand, tumours too small to appreciably increase the cranial contents frequently produce papillitis.

2. Manz¹ assigned dropsy of the intersheath space of the optic nerve as the cause. This he considered to be due to admission of the cerebro-spinal fluid in cases of intracranial pressure, or increase of subarachnoid fluid. This theory is supported by Broadbent² and others.

3. Schmidt, however, found that a coloured injection passed from the sheath space into the lymphatics of the papilla at the lamina cribrosa ; and he considered the inflammation to be produced not

¹ *Deutsch. Arch. f. klin. Med.* vol. ix. 1871.

² *Trans. Ophth. Soc.* vol. i. p. 108.

alone by the pressure of the fluid in the intersheath space, but by its pressure in these lymphatic spaces.

4. Leber¹ considers the inflammation to be caused not at all by the pressure of the fluid in the sheath, but by the conveyance of *pathogenic material* in that fluid to the optic nerve at the back of the eye.

5. Hughlings Jackson considers the most plausible hypothesis to be that first proposed by Schweller—viz. that a cerebral tumour acts as a source of irritation which has a reflex influence through the *vaso-motor* nerves upon the optic disc, leading to its inflammation. This theory is rejected, however, by most writers, on the ground that we possess no anatomical knowledge of such nerves.

6. Galezowski believes that the inflammation is in all cases propagated by continuity of tissue. If Galezowski and his followers were correct, we should expect to have papillitis with intra-ocular and intra-orbital neoplasms, also with local inflammatory changes, as orbital cellulitis, &c., but such is exceptional. Moreover, tumours are essentially non-inflammatory new-growths. How is it that a glioma of the cerebellum is attended with papillitis? Is it by continuity of tissue? Does the tumour excite inflammation which courses to the optic nerve and papilla without producing any visible organic change between its commencement and its termination?

Lawford and Edmunds have examined microscopically the meninges and optic nerves of a large number of cases of optic neuritis due to intracranial disease, and have in all of them found evidences of the presence of basal meningitis. Many cases have now been recorded of a trephining operation relieving the papillitis, although the tumour has not been removed. In some cases, indeed, the papilla has regained its normal appearance, although the cerebral substance has subsequently bulged through the wound, owing to the continued growth of the tumour.

It seems probable that these two factors, intracranial pressure and basal meningitis, are the chief ones concerned in the production of papillitis; but which predominates is one of the many pathological problems which have yet to be solved.

The *diagnosis* of papillitis is usually quite easy, as its signs are very definite, but occasionally cases are seen about which there may be considerable doubt. A condition, which has been termed 'spurious optic neuritis,' has been recorded where the disc has all the appearances of papillitis; no alteration occurs, however, even after several years. In high hypermetropia, again,

¹ Discussion at International Medical Congress, London, 1881.

there may be marked swelling and hyperæmia of the disc, with tortuosity of the vessels and indistinctness of the disc margin ; it may be impossible to state definitely that an early stage of papillitis is not present ; the stationary character of the appearance, however, excludes inflammation. Fine opacities in cornea, lens, or vitreous, may make the edge of the disc appear blurred ; careful ophthalmoscopic examination is all that is needed for the differential diagnosis.

The *diagnostic indications* of papillitis are of great importance. The three symptoms, headache, vomiting, and optic neuritis, when found together, are extremely suggestive of intracranial disease. They are, however, also found in uræmia, and in all forms of anæmia ; a careful examination, therefore, of the whole of the nervous system, together with a urine analysis and blood analysis, should be made in all cases.

The *prognosis* of papillitis is, in all cases, grave, and naturally will depend to a great extent on the cause. Many cases of papillitis with total blindness have been recorded where a large amount of useful sight has been regained. The most favourable cases are those of a syphilitic etiology which come under treatment early.

Treatment must be directed as far as possible to the removal of the cause of the affection. The various intracranial diseases must therefore be treated by appropriate measures independently of the papillitis, which, although serious on account of its pernicious effects upon the vision, is still only a symptom. The same rule applies to other causes. When no satisfactory cause can be found for the existence of papillitis, the use of mercurials, especially by the method of inunction, and of iodide of potassium, is advisable. Diaphoretic measures are often of great use, such as pilocarpine injections, hot-air baths, and sodium salicylate by the mouth. Leeches applied over the mastoid process often seem to do good. Neutral-tinted glasses should be worn, and the eye completely rested, a result to be obtained by the use of atropine. When medicinal treatment fails to subdue a papillitis, and even though no symptoms are present to localise an intracranial growth, it may be justifiable to trephine, and so relieve any existing high intracranial pressure.

Papillitis is occasionally met with in young girls from fifteen to twenty, and the cause usually assigned is some irregularity of the menstrual function; often, however, careful inquiry fails to elicit any history of this. The neuritis is generally preceded by severe headaches, and the prognosis as regards sight is extremely unfavourable.

ATROPHY OF THE OPTIC NERVE.

Etiology and Pathology.—The causes of optic-nerve atrophy are numerous, and may be divided into the following two great classes:

A. Atrophy not preceded by papillitis, or *simple* atrophy.

(a) Primary—(i) following retro-bulbar neuritis;
(ii) a primary degeneration.

(b) Secondary—(i) following a lesion of the optic nerve, optic chiasma, optic tract, or cerebral centre;
(ii) following a primary retinal degeneration.

B. Atrophy preceded by papillitis, or *consecutive* atrophy.

Primary simple atrophy often comes on without any apparent cause, and it is usually difficult to decide whether a given case is a primary degeneration or a sequela of a retro-bulbar neuritis. The causes of this latter disease have already been discussed, and may all produce optic atrophy; and now we must consider what may produce a primary degeneration of the optic nerve.

Tabes dorsalis is the commonest cause of primary optic degeneration, the frequency being variously stated to be between 13 and 42 per cent. It frequently occurs some years before the advent of ataxic symptoms, which are usually less marked if the eyes are affected than when there are no ocular symptoms. It is also found in disseminated sclerosis (but see Retro-bulbar Neuritis), general paralysis of the insane, hydrocephalus, and, rarely, in ataxic paraplegia. Diabetes has optic atrophy as an occasional symptom. A very important and interesting variety of primary optic atrophy is the so-called *hereditary optic atrophy*, or *Leber's disease*. This will be described below (see p. 275).

Plate X.

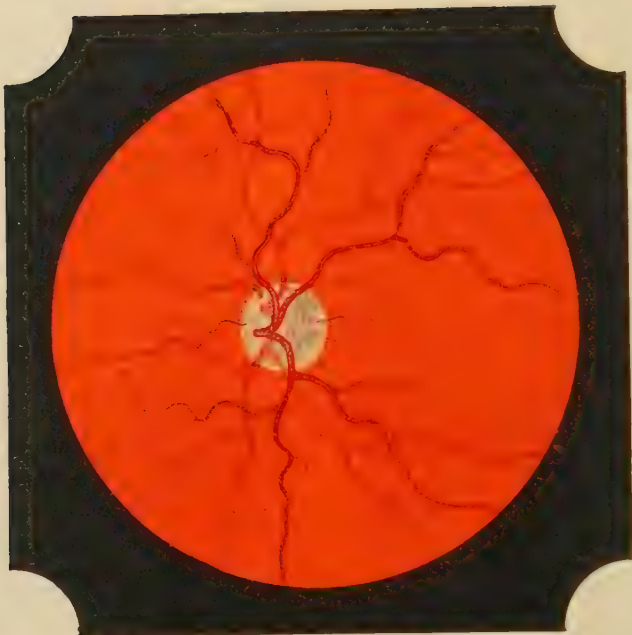


Fig. 1.—Primary Atrophy.

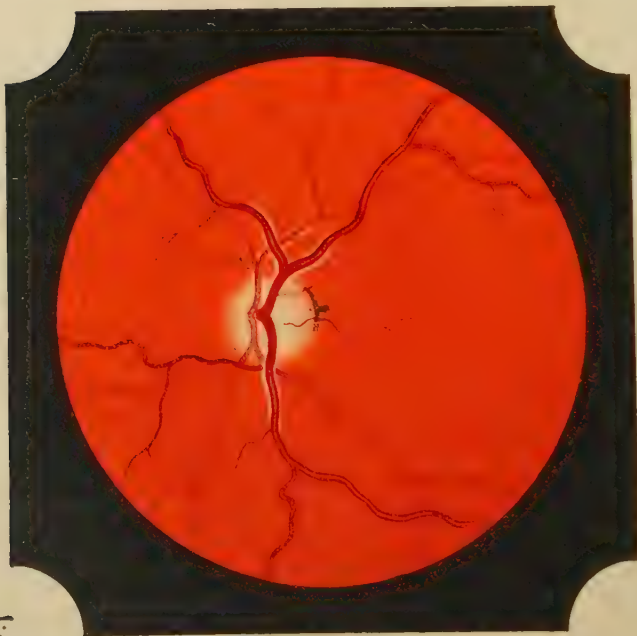


Fig. 2.—Consecutive Atrophy.

W.J.A.

Secondary simple atrophy is the result of some lesion, either of a portion of the brain (cerebral centre) from which the optic nerves arise, or of some part of the optic-nerve fibres between this centre and the eyeball (see Hemianopsia, p. 330). Pressure on the optic chiasma from various causes, lesions affecting the optic nerve in the skull and in the orbit, and blows upon the head (see p. 638), may all induce secondary atrophy. Again, as retinitis pigmentosa develops, a peculiar-looking simple atrophy of the optic nerve is produced (see p. 291).

Consecutive or post-papillitic optic atrophy to a greater or less extent succeeds all cases of papillitis.

Microscopical examination of the atrophied nerves in all forms of optic atrophy shows that the atrophy is not confined to the optic disc. The fibres and the connective-tissue elements of the nerve-trunks present various degrees of degeneration. As a rule, these elements are increased and the nerve-fibres partially or totally destroyed. In some cases the latter appear to be partially replaced by particles of fatty matter.

Symptoms.—1. *Pallor of the optic disc* is one of the first signs of atrophy of the optic nerve (see figs. 1 and 2, opposite p. 271); the usual slight rose or pink tint has become diminished or is altogether lost. The direct method of examining with the ophthalmoscope is the best here, and the details of the papilla can often be best seen when a feeble illumination is used. The various appearances of the healthy disc (see p. 242) should be borne in mind when making the examination; and it must not be forgotten that, as before mentioned, a very white-looking disc occasionally occurs in a perfectly normal eye. As a rule, however, the pallor of the disc is in proportion to the amount of atrophy present.

The appearance of the disc depends on whether the atrophy is simple or consecutive, and it is largely by carefully noting these points of difference that the diagnosis between the two forms of optic atrophy is made. These differences are the following:

(a) The colour of the disc in simple atrophy is grey or white. Frequently only a sector of the surface is discoloured, the

position of the atrophied portion being downwards and outwards. At the bottom of the physiological cup the lamina cribrosa is usually very evident. In consecutive atrophy, on the other hand, the disc is of a woolly-white colour, and the lamina cribrosa is generally invisible, though after a long interval it may again come into view.

(b) The *outline* of the disc in simple atrophy is always very clearly defined, the edges being extremely well cut; while in consecutive atrophy the reverse is always the case, the edges being blurred and irregular in one or more places. This difference is of extreme importance in the differential diagnosis between the two forms.

(c) The appearance of the *vessels* varies considerably. In simple atrophy they may be either normal or somewhat contracted. Rarely is the contraction extreme, the vessels in cases of optic atrophy with retinitis pigmentosa being an exception. In early cases of consecutive atrophy the veins are usually distended and tortuous, but later both they and the arteries are contracted, and consequently closely resemble the vessels in simple atrophy. Frequently, however, in consecutive atrophy the vessels have white lines along them, and when present these are a very valuable diagnostic sign, as they are never found in simple atrophy.

2. *Diminution of visual acuity* almost invariably takes place from the onset of the affection. Its rate of progress is also subject to variation: as a rule, it proceeds slowly towards total blindness, but it may become more rapid and lead to this result in a comparatively short time; on the other hand, it occasionally becomes stationary.

3. *Impaired colour vision* is an almost constant symptom. The patient at first finds a difficulty in recognising green, and if asked to match a pure green with the confusion-colours for green (see Colour Vision) he will be unable to do so. Green appears to him to be grey or yellow. Further than this, the progress of the disease is marked by gradually increasing trouble in the perception of colours. Next the red, and then the yellow, can no longer be recognised with any degree of certainty, thus leaving only the power of discerning blue. Finally, this also disappears, and the colour-blindness is complete.

4. *Alterations in the visual field.*—The failure of visual acuity, already mentioned, is usually accompanied by more or less *contraction of the visual field for white*. This generally consists in a regular contraction, the outline of which is concentric with the macula; it may, however, take the form of a sector-like defect, or one-half of the field (apart from the hemianopsia of cerebral disease) may be lost; lastly, the alteration may consist in an irregular scotoma in the middle of the field. Again, it is frequently found that *the limits of the field for colours are also contracted*. By the method

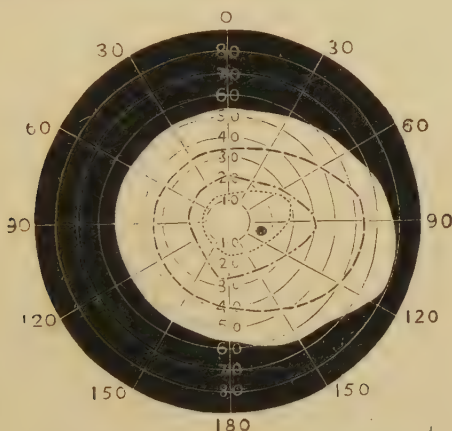


FIG. 60.—Visual Field in Optic Atrophy.

— — — blue, red, green.

of testing indicated in the chapter on Perimetry, we find that the field for green becomes smaller by degrees, and finally disappears. With the progress of the atrophy this contraction of the field for green is followed by a similar limitation for red, then for yellow, and finally for blue. Fig. 58 shows the commencing concentric contraction of the visual field; the contraction for colours is relatively more marked than that for white. The contraction of the field for colours is, in fact, more constantly found than that for white.

5. The *direct pupillary light reflex* is completely lost in well-marked cases of optic atrophy, the pupil being dilated and fixed. The dilatation is less than that produced by

atropine applied locally. In slight cases of optic atrophy, hippus is present, and the pupillary reflex resembles that found in early cases of retro-bulbar neuritis (see p. 260).

6. *Nystagmus* is frequently found in cases of optic atrophy, and especially if the subjects are young.

Prognosis is always unfavourable, especially in cases where the cause of the affection is beyond control. Progressive atrophy usually attacks both eyes, and terminates very often in complete blindness. Perimetric observation of the visual field at stated intervals gives the best indication of the progress of the disease. Those forms in which the visual field is not concentrically contracted, but diminished in one part more than another, are the least pernicious.

Treatment is frequently of no benefit.

The *continuous voltaic current* has been tried by Remak, Benedict, Pye-Smith, Gunn, and others, with the effect of some restoration of vision. The current must be varied in strength according to the susceptibilities of the patient. About 3 to 5 milliampères can usually be borne; but it is well to be very cautious in the application of this remedy, as a comparatively weak current sometimes produces vertigo and other symptoms, which are very alarming to the patient. The positive pole is placed over the mastoid process, and the negative pole upon the closed eyelids. The current is continued for five minutes at each sitting, and is repeated daily. Gowers states that he has tried this treatment in many cases, but without results which could reasonably be ascribed to the treatment.

The *hypodermic injection of strychnine* is advocated by Nagel and others. He injects about 1 milligramme of the sulphate of strychnine, dissolved in 10 minims of distilled water, every second day. If there is no improvement by the end of six weeks, the treatment can be discontinued as useless.

General treatment must vary according to the evident or probable cause of the affection—*e.g.* the removal of toxic influences, abstinence from excesses of all kinds, &c. Where syphilis is suspected, the appropriate treatment for this disease should be carried out.

Hereditary Optic Atrophy, or Leber's Disease.

This is a rare form of atrophy of the optic nerve, and, as a rule, comes on between the ages of twenty and thirty-five in several male members of the same family. It is usually transmitted through the females, who themselves are rarely affected. There is frequently a history of more or less excessive tobacco smoking. Endeavours have been made to trace connection between it and masturbation. There is often an epileptic family history. The disease is progressive, though rarely does complete blindness result. At the outset there is often slight papillitis, and in all cases a preceding retro-bulbar neuritis is probably present. No form of treatment has been able to arrest the disease.

Hæmorrhages into the Optic-Nerve Sheath.

Hæmorrhages into the optic-nerve sheath are considered to take place, but they have never been proved to exist. A few cases have been recorded where they have been thought to be present. In these cases sudden blindness has occurred, and ophthalmoscopic examination has shown a retinal œdema, a cherry-coloured spot at the macula, and either normal or slightly contracted vessels, with no hæmorrhages. Pulsation occurs on pressure of the globe. The subjects of these symptoms have usually been girls suffering from amenorrhœa. Optic-nerve atrophy generally ensues. There is also the traumatic form (see p. 638).

DISEASES OF THE RETINAL VESSELS.

Ischæmia of the retina signifies a sudden, often total, arrest of the retinal blood-current, accompanied by entire loss of sight. It is unattended by any tissue-change of the retina and optic nerve. Both eyes are usually affected.

Symptoms.—The optic disc is pale or white. The arteries are either completely empty and reduced to fine white threads, or they may contain a delicate continuous column of blood, which is seen as a red line in the axis of each vessel, or they may be empty in certain parts and contain a little blood in other parts. The veins are generally smaller than normal,

and may be more contracted in one part than another. The affection is very rare. It is said to be sometimes present during an epileptic seizure. Cases have been recorded where repeated transient attacks of blindness have occurred for several years. These attacks may last only a few minutes, or even seconds, and in the intervals the sight completely returns. Ophthalmoscopic examination during an attack has shown ischæmia of the retina, usually however confined to a portion, and not involving the whole of the fundus. The cause of the condition is not definitely known. After many attacks, and corresponding recoveries, one may occur resulting in permanent blindness, either total or involving only a part of the visual field; the appearance is then exactly similar to embolism or thrombosis of the central artery.

Embolism of the central artery of the retina may occur in the trunk or its branches, and may be complete or partial. The clot is usually just behind the lamina cribrosa. It is very rarely bilateral.

Symptoms.—Sudden unilateral blindness occurs, which may have been preceded by temporary obscurations. Supposing the trunk of the artery to be affected, we find by the ophthalmoscope that the arteries are extremely shrunken, and their smaller branches invisible. The veins also are reduced in size, but more so at the optic disc than in the rest of their course. Sometimes a broken column of blood can be seen in the veins, and then during the first few days an oscillatory movement of the blood may be observed. Pressure upon the globe will not produce pulsation either of the arteries or the veins. Hæmorrhages are few and slight. The characteristic feature is a greyish-white opacity surrounding the region of the macula; this is several times the diameter of the optic disc in breadth, and is marked at its centre by a *cherry-red spot* corresponding to the position of the fovea centralis. A similar white haze often surrounds the papilla. The brilliancy of the red spot at the fovea is not of equal intensity in all cases; sometimes it is speckled with grey; usually, however, it is of a bright cherry-red colour, and is either circular or oval. Several theories as to the cause of its red hue have been suggested, but it is probably pro-

duced by contrast between the white haze of the surrounding retina and the red colour of the blood in the choroid being seen through the thin fovea centralis. In some cases, however, it may be due to a hæmorrhage into the retina or choroid; while Nettleship considers a central choroido-retinitis as its cause. In a *post-mortem* examination, Nuel found a marked injection of the blood-vessels in the macular region. Occasionally, however, although the main trunk is occluded, the circulation in a small sector of the fundus may remain, and consequently some vision. Either a cilio-retinal vessel is present, or one or more branches of the central retinal artery leave the main trunk behind the embolus. When this is the case, it is usually the macular branches which are not occluded, and consequently central vision is not wholly lost.

Some days after the attack there is frequently re-establishment of the circulation, but the retina has by this time become atrophic. This re-established circulation may be due to the passing away of spasm of the arterial walls, the increase in the diameter of the lumen allowing blood to pass between the walls and the clot; or it may be that the embolism itself shrinks and allows some blood to pass it.

After some weeks the retina becomes again clear, and the optic nerve takes on the white appearance of atrophy. The cherry-red spot at the fovea is then less marked; there are generally a few specks and traces of deposit in the retina.

As a rule, there is no sight at any time, although a few cases are recorded in which some perception of light has re-appeared after a short time in the outer part of the field, due possibly to some anastomosis with the choroidal vessels in the neighbourhood of the ora serrata.

Fig. 1, opposite p. 219, copied from Liebreich's atlas, represents the appearance of this affection. The opacity of the retina in the region of the macula and of the optic disc is often more marked than this, and the veins less visible. *If a branch only* of the retinal artery is obstructed, the cloudy opacity is localised, and only the corresponding part of the retina suffers. This is indicated by a scotoma, which may vary in extent from a mere spot to half the visual field.

The *causes* are chiefly cardiac valvular diseases and endocarditis. It is also, more rarely, caused by Bright's disease and advanced pregnancy.

The *prognosis* is very bad.

Treatment.—Very little can be done. If seen immediately after the occlusion has taken place, attempts may be made to dislodge the embolus. The eyeball should be massaged, and a paracentesis of the anterior chamber has occasionally had some effect. Inhalations of amyl nitrite may be tried.

Thrombosis of the central artery of the retina resembles very closely, in its symptoms, embolism of the central artery, and the diagnosis from it is often difficult. There are, however, often premonitory symptoms, such as headaches, giddiness, and transient temporary blindness, which are usually absent in embolism. The walls of the blood-vessels show the typical appearances of arterio-sclerosis, the so-called silver-wire appearance. In thrombosis there is an absence of cardiac and renal symptoms. Like embolism, it may involve the whole or only a branch of the central artery. Its causes are the causes of arterio-sclerosis in general. If of a rheumatic origin, salicylate of sodium should be administered internally. Alternate local application of atropine and eserine has been tried, as well as inhalations of amyl nitrite, with the hope of driving the thrombus forwards.

Thrombosis of the central vein of the retina may be total or only partial. There is great distension of the veins, and, as a whole, profound hæmorrhage, so much so that details of the fundus are, to a large extent, hidden. Occasionally the blood may break through into the vitreous. The arteries, if visible, will be seen to be greatly contracted. The disease tends to recur, and finally, if not from the first, all sight is lost. The cause is usually some cardiac disease, or general arterio-sclerosis, albuminuria being frequently present. It is almost confined to elderly people, but is not unknown in children suffering from inherited syphilis. Treatment must be on general lines.

Symmetrical Macular Changes in Infantile Cerebral Degeneration.—This rare and fatal disease has, in its early stages, certain well-marked and characteristic ocular signs. In the

macular region there is a greyish patch, rather larger than the disc, with a cherry-red spot at its centre, reminding one forcibly of the appearance found in embolism of the central artery of the retina. The rest of the fundus looks normal, but in a later stage of the disease definite optic atrophy sets in, with all the characteristics of the primary form, the child being completely blind. Pathologically, it is found that the retinal changes are confined to the outer molecular layer, the tissue of which is spaced out. The optic nerve shows an increase of interstitial fibrous tissue, together with a number of round cells. The cause of the disease is unknown; it is certainly not embolic. No treatment has been found of any avail, the child usually dying within two years of the onset of the disease.

Retinal hæmorrhages may occur without inflammation. The number, aspect, and extent of these extravasations vary indefinitely; they may be divided into superficial and deep varieties. The superficial naturally occur in the course of the vessels in the nerve-fibre layer, and hence present a striated aspect. The deeper extravasations of blood pass backwards between the fibres of Müller; they are not striated, but are seen as irregular rounded masses; they vary in volume and depth, but usually occupy only the intergranular layer. Occasionally the blood passes forwards into the vitreous body, or backwards between the retina and choroid. *Hæmorrhage in the region of the yellow spot* deserves special mention on account of its frequency and importance. This is a rounded or elliptical patch of varied extent; it is usually about three or four times the size of the optic disc. Smaller hæmorrhagic points are often seen in its neighbourhood. The retina is never raised, and the extravasation is never deep. The absence of nerve-fibres and of any considerable vessels in this region explains these peculiarities. The frequency of return of visual acuity also shows the slightness of the lesion as regards the cones and the ganglion-cells; indeed, it is possible that the blood has not extravasated within the yellow spot, but from some marginal vessel, and that it has filtered between the retina and the vitreous body, forming a *subhyaloid* or *pre-retinal hæmorrhage*. This form takes on a boat-shape, with

an upper margin remaining horizontal in all positions of the head. If resorption occurs, the clot becomes decolorised centripetally. If the resorption be incomplete, white patches remain, mixed with more or less pigmented matter. This is after large or repeated hæmorrhages. Occasionally pigmentary striæ originate from a previous hæmorrhage, owing to changes in the blood-pigment—the so-called *angioid streaks*. They lie beneath the retinal vessels. When the macula is affected the central vision is suddenly impaired or lost. This may not be an absolute central scotoma, but is often a uniform cloud, covering objects in front of the eye. If the hæmorrhage is peripheral, the visual field is affected accordingly.

The *causes* of retinal hæmorrhages may be classified as follows:

1. *Injuries*, such as blows, wounds of the eye, causing sudden alteration of the intraocular tension, as when there is escape of aqueous or vitreous humour.

2. *Derangements of the vascular system.*

General arterial sclerosis.

Heart-disease, especially mitral.

Embolism and thrombosis of small arteries.

Miliary aneurysms.

Fatty degeneration (after endarteritis).

3. *Alteration in the quality of the blood.*

Diabetes.

Albuminuria.

Diminished blood-pressure due to a sudden and great loss of blood.

Leucocythæmia.

Pernicious anæmia; never in chlorosis.

Purpura and scurvy.

Hæmorrhagic diathesis.

Typhoid fever—usually during the third week—synchronous with intestinal hæmorrhage.

Pyæmia and septicæmia.

Malaria.

Cirrhosis of liver.

4. *Local diseases.*

Optic neuritis.

Retinitis.

Thrombosis of central retinal vein.

Embolism of central retinal artery.

Myopia (progressive):

Some of these conditions also give rise to inflammatory changes, and will be referred to again later. Retinal hæmorrhage occasionally occurs in young persons. It is usually central, extensive, and relapsing. A subhyaloid or vitreous hæmorrhage is often found in these cases, the subjects of which are generally feeble or anæmic; they are also frequently myopic. Inherited syphilis is probably a factor in the causation.

Angioid Streaks in the Retina.—This, a very rare condition, is probably the result of hæmorrhages in the deeper layers of the retina. Pigmentary changes in these hæmorrhages take place, leaving striæ scattered, as a rule, over a considerable area of the fundus.

The Retina in General Arterio-sclerosis.—Owing to the fact that changes in the walls of the retinal vessels are readily observed by means of the ophthalmoscope, general arterio-sclerosis may be frequently diagnosed, though other evidence of its presence may at the time be wanting. A hyaline degeneration of the vessel walls affects chiefly the adventitia and intima, and is recognised by certain well-marked signs, described by Marcus Gunn: ¹

1. Irregularity in the breadth of the arteries.
2. Tortuosity of the arteries.
3. A narrow and very bright central light reflex.
4. Loss of translucency of the arteries. This produces in the veins an appearance of interruption when crossed by the arteries.
5. Irregular dilatation of the veins. Where a vein is crossed by an artery, pressure is exerted upon it, causing peripheral dilatation.
6. Later changes may occur, such as œdema of the retina, and retinal hæmorrhages.

¹ *Trans. Ophth. Soc.* 1898, p. 356.

The etiology of the condition is the etiology of general arterio-sclerosis, and so need not be here considered. Suffice it to say that a discovery of these well-marked retinal signs should lead to a grave prognosis, renal disease or cerebral hæmorrhage being the almost inevitable result.

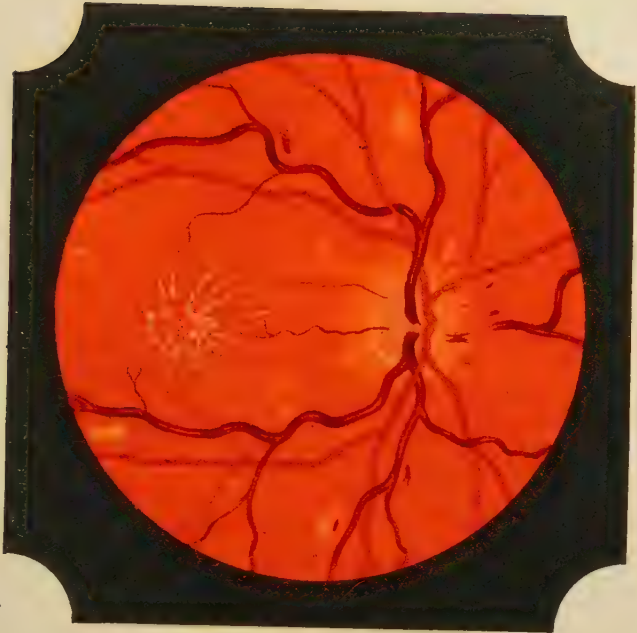
RETINITIS.

Inflammation of the retina seldom occurs idiopathically; it is usually the result of some constitutional dyscrasia, as albuminuria, glycosuria, syphilis; or else it is caused by extension of an inflammation from the neighbouring choroid or ciliary processes. We shall consider retinitis under three chief headings:

1. *Albuminuric retinitis* and its allied forms occurring in glycosuria, leucocythæmia, &c.
2. *Syphilitic retinitis*.
3. *Pigmentary retinitis*.

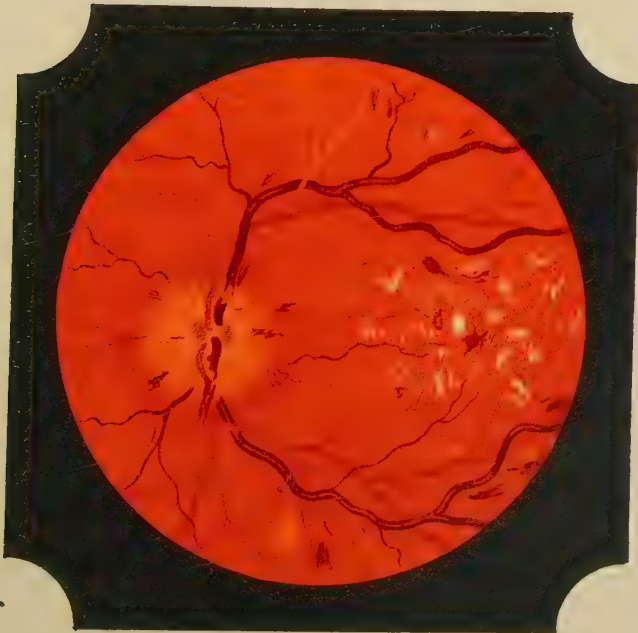
Albuminuric Retinitis.—*Ophthalmoscopic signs*.—In the early stage of the affection we find a dull grey haze all over the central region of the fundus. The papilla is somewhat swollen and its outline blurred. There are generally some hæmorrhages in the region of the disc, and a few *soft-edged* white patches can be seen in various parts of the retina. After a few weeks, when the affection is established, we find—(1) *White glistening spots or patches*, sometimes as small as a pin's head, more or less collected into groups around the yellow spot (see fig. 1, on opposite page). Sometimes they assume the form of white or yellowish-white striæ, arranged in a radiating manner around the same focus. Larger spots than these of the macular region are found scattered over the fundus; they are larger, less glistening, and their outline is less defined; when occurring in the vicinity of a vessel, they are usually found to cover it. (2) *Hæmorrhages* are, as a rule, found; these may be small and in the form of dots, but they are generally striated and torchlike in appearance, being placed in the layer of optic-nerve fibres. They vary in colour according to the length of time which may have elapsed since their extravasation, the most recent being of a bright arterial red colour, whilst the oldest are of a yellowish-white, waxy appear-

Plate XI.



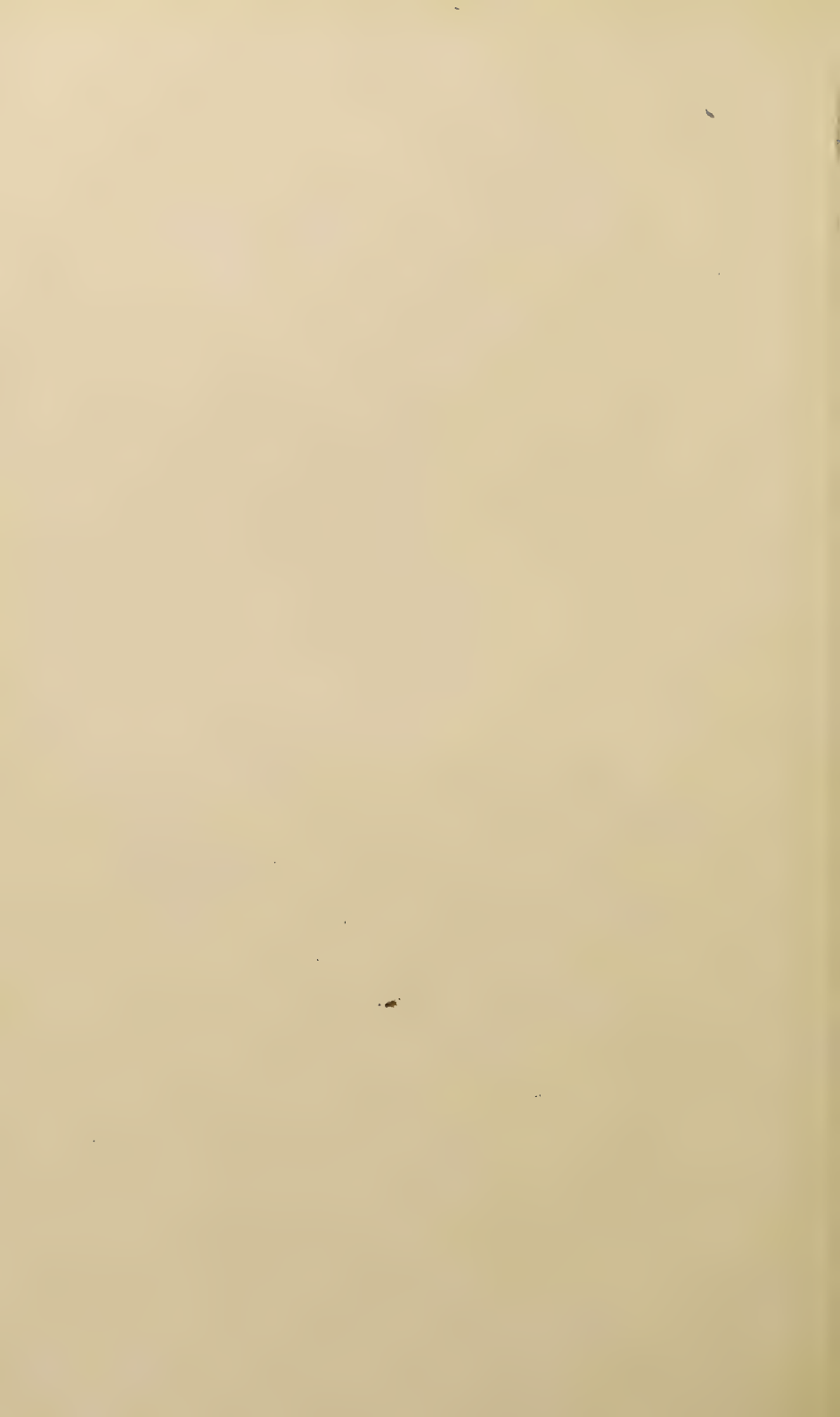
W.J.R.

Fig. 1.—Albuminuric Retinitis.



W.J.R.

Fig. 2.—Diabetic Retinitis.



ance. As a rule they run parallel with the larger vessels, although the particular vessel from which the blood is extravasated can seldom be seen. When large they are irregular in shape, and extend to the deeper layers of the retina. (3) The *optic papilla* may be only slightly affected, but is usually swollen, hazy, and blurred in outline. In occasional cases there is an inflammatory change in the perivascular lymph space which gives to the arteries or veins a whitish halo or even an opaque white appearance along the whole or a part of their course, through which the contained blood can be dimly seen. (4) *Detachment of the retina* sometimes occurs, but it is not common. The detachment is usually of the serous form, and only occasionally hæmorrhagic. It may be bilateral, and is commoner in the albuminuric retinitis of pregnancy than in the other forms. In most cases we find that one or other of these changes predominates, and, according to the most conspicuous feature, Gowers¹ proposes to distinguish four types of cases—the degenerative, the hæmorrhagic, the inflammatory, and the neuritic.

Both eyes are usually affected, but the lesion is almost always more marked in the one eye than in the other; occasionally the disease is unilateral. This unilateral form is generally of the hæmorrhagic type, and perhaps indicates that only one kidney is diseased. It is less grave than the bilateral form.

Functional disturbances do not always correspond with the ophthalmoscopic signs. It is not uncommon to find considerable retinal disturbance with only slight amblyopia; and on the other hand, the retina may appear to be but slightly affected, whilst the patient can hardly see sufficiently to find his way about. Transient attacks of blindness are, indeed, a common premonitory symptom of the disease, and are especially met with after uræmic convulsions. If the patient complains of defective vision, the condition is probably far advanced. The gravity of the functional disturbance depends greatly upon the region affected; so long as the yellow-spot region remains intact the visual acuity is tolerably good, but as soon as this part is attacked the central vision immediately suffers.

Diagnosis.—The ophthalmoscopic signs above described

¹ *Medical Ophthalmoscopy*, p. 185.

are by no means pathognomonic of this disease, since they are very similar in many cases of intracranial tumour, especially cerebellar, and in the various forms of anæmia. Retinal hæmorrhages have an extensive etiology (see p. 280), and glistening macular spots are also present in diabetes. It is the condition of the cardio-vascular system, the high arterial tension as evidenced by the high-tension pulse, the accentuated aortic second heart-sound and the reduplication of the first sound at the heart's apex, and the presence of renal disease as shown by the low specific gravity of the urine, the small percentage of urea, and the presence of casts and albumen, which confirm the diagnosis suggested by the ophthalmoscopic signs.

Prognosis.—The relation between the progress of the kidney-affection and that of the retinitis is not constant. With the improvement of the renal disease there is usually a tendency towards subsidence of the swelling, absorption of the deposits and extravasations, and recovery of vision. This is particularly exemplified in the albuminuric retinitis of pregnancy. The lesion of the kidney may remain stationary or become aggravated, whilst that of the retina may disappear, and vision be re-established. Even where the retinal deposits persist, there is sometimes a very considerable improvement in vision. On the other hand, the urine may be almost free from albumen, but the retinal affection gets worse and worse. As a rule, in the milder forms of albuminuria, the lesions of the retina disappear, and the sight is restored; but in the severer cases, where there has been swelling of the optic disc and œdema of the retina, the loss of vision is very great, and is not likely to improve, although it may remain stationary. Should severe atrophic changes of the optic nerve supervene, the sight may be permanently reduced to an extreme degree ($V < \frac{6}{60}$, J 20, or even fingers only).

The general prognosis of albuminuric retinitis is very grave, since its presence indicates an advanced condition of the renal disease. The more marked the retinal hæmorrhages, the more grave must be the prognosis.

In all cases where albuminuric retinitis is suspected, the urine should be *repeatedly* examined, the absence of albumen on one occasion being insufficient to disprove the existence of

renal disease. Not only should the urine be tested for albumen, but the percentage of urea present should be determined and an examination for casts performed. Poverty of urea and presence of casts denote renal disease, whether albumen be found or not.

Etiology and pathology.—Albuminuric retinitis is most commonly found in the advanced form of chronic interstitial nephritis, although it is not a very frequent complication of that affection (probably not more than 8 or 10 per cent.). It is occasionally found in chronic parenchymatous nephritis, but an interstitial fibrosis accompanying this may be the determining cause. Acute parenchymatous nephritis and amyloid disease of the kidney probably never produce retinitis. It occurs in the albuminuria of pregnancy, and occasionally follows scarlet fever in children; but in both these instances the kidneys may have been unhealthy previously. Chronic interstitial nephritis is not unknown in children, and several cases of this disease accompanied by albuminuric retinitis are on record. The chief factor in the causation of albuminuric retinitis is, probably, high arterial tension.

On *microscopic examination* of the affected optic disc and retina, we find the following changes: (1) The nerve-fibre layer of the retina is swollen and oedematous; there is also oedema in the outer molecular layer in some cases. (2) The *arteries of the retina* present thickened patches in certain parts of their course; and sections through these nodules show a general thickening of all their coats, especially of the sub-endothelial part of the intima, in consequence of which the outside diameter of each vessel is much increased, and its lumen diminished or entirely obliterated; indeed, according to Brailey and Edmunds,¹ some impervious arteries are generally to be found in a state of fibrous or hyaline degeneration. The *capillaries* also present a marked degree of hyaline thickening; although thickened, they are nevertheless disposed to rupture, and this is probably the source of the hæmorrhages. (3) *Blood-corpuscles* are found more abundantly in the region of these thickened patches than in other parts of the retina; they are found not only in the inner layers of the retina, where

¹ Vide *Ophth. Trans.* vol. i. p. 45.

the capillaries exist, but also in the intergranular layer. After a time the hæmorrhages thus extravasated are seen as *crystalline masses* and *fatty substances*. (4) *Inflammatory nuclei*, probably of the neuroglia, are found in the inner layers of the retina, also large spheroidal cells in an advanced stage of fatty degeneration (corpuscles of Gluge). (5) The *fibres of Müller* are greatly thickened, and separated by sero-albuminous fluid; they eventually undergo fatty degeneration, and in all probability give rise to the above-mentioned *granular spheroidal cells*. (6) The *optic nerve* shows, in every instance, signs of active neuritis. The degree varies with the type the disease follows. In some forms the disc will be considerably swollen, and the papillitis more marked than the retinitis; while in other cases the retinitis will predominate.

The glistening white patches in the macular region are composed of minute granules of fat, the result of degenerative changes in the retinal tissue, both cells and fibres, and in the inflammatory exudation; they probably are never absorbed. The radiate arrangement is produced by folds in the œdematous retina. The more woolly, less glistening patches are due to effusion or œdema. These patches may become absorbed.

Treatment.—The general treatment must be directed to the renal affection. Locally, the use of smoked glasses, and rest to the eyes, are all that can be advised.

Diabetic retinitis is very rare, and is so similar to the albuminuric form that it is almost impossible to distinguish the one from the other by means of the ophthalmoscope alone. The white spots, however, in the macular region are larger and more irregular in outline in this affection; they have no tendency to be radially arranged around the yellow spot, and they rarely become confluent. Hæmorrhages occur in all parts of the fundus; the extravasations appear either in the form of dots, or as irregular patches or lines. In the early stage papillitis is not a marked sign, but later it becomes very pronounced. It is uncommon to find œdema of the retina. The disease tends to progress, *new vascular formations appear in the vitreous*, and the latter becomes crowded with membranous and other opacities; lastly, the retina becomes detached, with complete loss of vision. The retinitis is

symmetrical. The prognosis is very unfavourable. The treatment must be entirely directed to the diabetes. Local blood-letting, by leeching or other means, blisters, scarifications, &c., are more likely to do harm than good. Diabetic retinitis may be preceded by diabetic amblyopia.

Leucocythæmic retinitis was first noticed and described by Liebreich. It is characterised by the existence of yellowish rounded hæmorrhagic spots or patches; these occur in the region of the macula, and at the periphery of the fundus; they are perceptibly prominent, and, when examined by the direct method, they may be seen to project into the vitreous cavity. In the majority of cases, whitish streaks can also be seen along the course of the retinal vessels. Various scotomata, corresponding to the position of the whitish patches, are found to exist in the visual field. The normal orange-red colour of the whole fundus is frequently changed to that of a paler orange-yellow. The spots and streaks are due to accumulations of leucocytes which have escaped from the walls of the vessels by diapedesis, and the change of tint of the whole fundus is caused by the altered condition of the blood in this disease (O. Becker).

This affection is by no means constant in leucocythæmia; it occurs only in from 20 to 30 per cent. of the cases, and these are mainly in the splenic form. Treatment must be general.

Well-marked retinitis may occur in many cases of pernicious anæmia. Hæmorrhages with white patches are found, and occasionally papillitis may also be present. Neuro-retinitis has also been found in chlorosis.

Syphilitic Retinitis is mostly associated with, and secondary to, choroiditis. A description of *syphilitic choroido-retinitis* will be found on p. 202. Occasionally, however, we meet with isolated syphilitic retinitis.

Symptoms.—*Ophthalmoscopic examination* shows a *cloudy opacity*; this may be confined to the region of the yellow spot and optic disc, or may extend over a larger area of the fundus, or it may follow the course of the larger retinal vessels in the form of cloudy streaks. The periphery of the retina is usually clear and visible. Occasionally the disc is swollen. Hæmorrhages are very rare. Very often, as in choroido-retinitis, we

find numerous fine 'dustlike' opacities situated in the deeper portions of the *vitreous*, near the posterior pole of the eye. This *vitreous haze* is apt to be mistaken for optic neuritis or neuro-retinitis, unless care is taken to use the plane mirror as in retinoscopy. Larger floating opacities of the vitreous are also common, and not unfrequently we may detect the signs of recent or old iritis in the pupil. The smallest traces of pigment upon the front of the lens, or of adhesion of the iris to the lens, are enough to establish this.

Failure of vision is very marked from the first, and may, if the case is left untreated, go on to complete blindness. This failure is often greater than the ophthalmoscopic changes would lead us to anticipate. The patient also complains of fog before the eyes, *muscæ volitantes*, and of inability to see in a dull light. There is always torpor of the retina, which often goes on to absolute *night-blindness*. This form of retinitis, as a rule, attacks one eye at a time, but, in the absence of proper treatment, it sooner or later comes on in the second eye. It is one of the secondary symptoms of syphilis, and usually appears between the sixth and eighteenth months after the primary affection. Its course is generally protracted over many months, and evinces a tendency to relapses and exacerbations after slight temporary improvements. When seen early and treated by mercurials, great benefit may be effected, but with neglect of treatment, and under bad hygienic conditions, it generally gets worse, and goes on to more or less complete atrophy of the optic nerve and retina.

Retinitis circinata is a rare form of retinitis, and of doubtful pathology. First described by Fuchs, it presents a well-defined ophthalmoscopic appearance. A somewhat dark and pigmented macular region is surrounded by a horseshoe-shaped ring of woolly white spots, some discrete, others confluent. The ring may, however, be complete, and its diameter is two or three times that of the disc. There is usually a part of the fundus lying between the macula and the disc, which appears normal. A few small hæmorrhages may be present, and a part of the retina may be cedematous. The retinal vessels pass uninterruptedly over the white ring and look normal. It seems to be a product of old age, and,

curiously, is much more common amongst women than men. A typical bilateral retinitis circinata has, however, been seen in a child only a few months old. Two views have been held as to the pathology of this disease. Fuchs originally considered the white wreath to be caused by fibrous exudation into the external retinal layers. It seems to be more probable, however, that a previous hæmorrhage has undergone fatty degeneration, or simply becomes decolorised. The condition is chronic, though cases have been recorded in which the white patches have slowly disappeared.

Retinitis Proliferans.—This rare condition is still very imperfectly understood, and its pathology is even now a matter of dispute. Its ophthalmoscopic signs are, on the contrary, well known. A shining, pure white, opaque substance is seen in the vitreous supplied by blood-vessels, which appear to be continuations of the retinal vessels. Occasionally small hæmorrhages have been seen upon it, and they frequently co-exist in the retina either of the same or of the opposite eye. Usually it appears to spring from the posterior pole of the eyeball, and is generally quite localised. Vision is not entirely lost, a central scotoma, corresponding to the opacity, being present, with fairly good peripheral vision. Pathologically, the condition may be explained as being the result of hæmorrhages into the retina and vitreous, with a new formation of connective tissue. It is doubtful whether any retinitis exists, or precedes the formation of the fibrous tissue; perhaps it should rather be considered to be a primary degenerative fibrosis of a toxic origin, allied to cirrhosis of the liver and sclerosis of the spinal cord. The etiology is obscure. In a few cases diabetes has been present. Probably chronic arterial high tension exists, which is perhaps due to chronic Bright's disease, or to a syphilitic endarteritis. It may follow hæmorrhagic glaucoma, and injury must be mentioned as a predisposing cause. Treatment must be on antiphlogistic lines, and attempts made to lower the general blood-pressure. Mercurial inunctions, potassium iodide internally, and pilocarpine injections have been of some benefit.

Differential Diagnosis between Choroiditis and Retinitis.—It is, for anatomical as well as physiological reasons, exceptional to

have inflammation of the choroid alone, since the outer layers of the retina are nourished entirely by the choroid. Consequently, as one would expect, inflammation of the choroid always involves the most important elements of the retina—the rods and cones: irritation at first—*phosphenes*—and other visual disturbances; destruction at last—*scotomata*. Retinitis, on the other hand, frequently occurs without inflammatory changes in the choroid. Thus choroido-retinitis is almost invariably secondary to choroiditis.

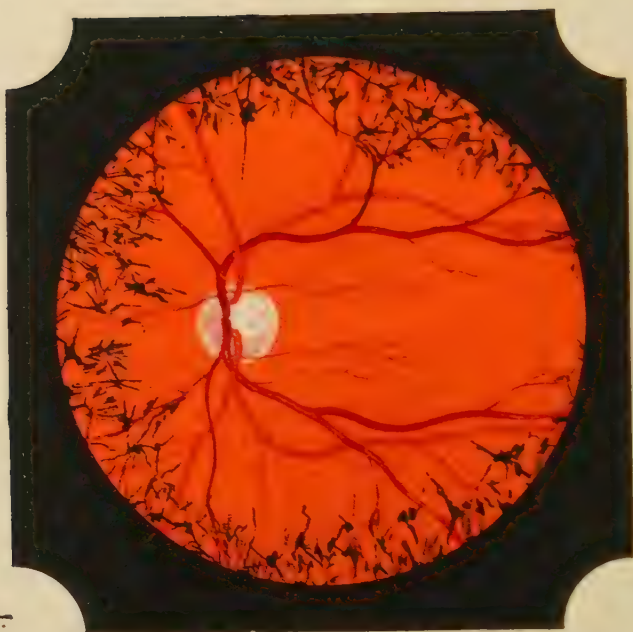
In distinguishing the two affections, the following points should be observed: In retinitis, there is usually *papillitis* with its characteristic features; the *changes are finer* in character, not so gross as in choroiditis; *hæmorrhages* frequently occur along the course of the vessels, and the *exudations* or pigmentary deposits may be situated *on* the retinal vessels, temporarily hiding them from view. In *choroiditis* (by choroiditis we mean inflammation of the choroid which originates and mainly involves that membrane) the changes occur *beneath* the retinal vessels; the latter are often seen coursing over the exudations or atrophic patches; *vitreous opacities* are usually present; the lesions are, in the atrophic stages, very pronounced; there are *no visible changes in the disc or retinal vessels*, neither are retinal hæmorrhages seen. In *choroido-retinitis*, in which both membranes are equally involved, a combination of the above ophthalmoscopic signs is found. Syphilitic choroido-retinitis is liable to simulate retinitis pigmentosa. For the points in diagnosis, see p. 293.

DEGENERATIVE CONDITIONS OF THE RETINA.

Retinitis Pigmentosa or Pigmentary Degeneration of the Retina.—The chief *symptoms* are: 1. Pigmentary deposits in the peripheral portions of the retina, and other ophthalmoscopic changes; 2. Night-blindness; 3. Concentric limitation of the visual field.

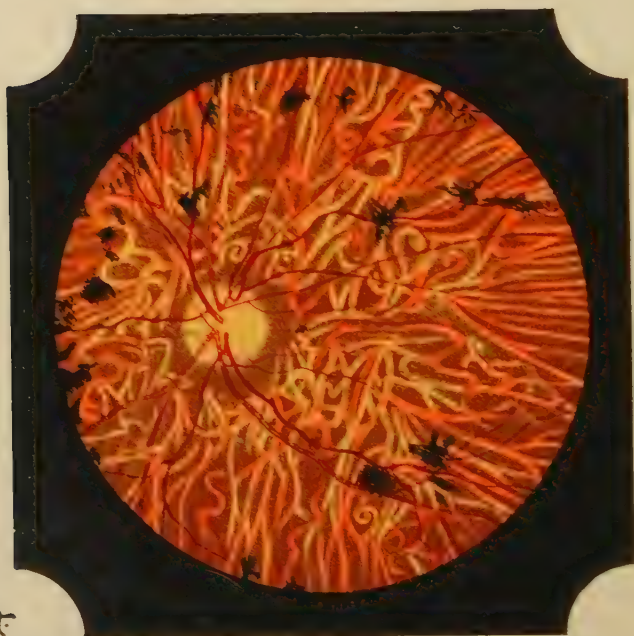
1. The *pigmentary deposits* in the retina may be easily overlooked in the earlier stages of the affection, inasmuch as the central portion of the fundus then appears quite normal. On examining the periphery of the retina, the appearance

Plate XII.



W.J.F.

Fig. 1.—Retinitis Pigmentosa (early stage).



W.J.F.

Fig. 2.—Retinitis Pigmentosa (advanced stage).

presented in fig. 1, on the opposite page, will be observed. The masses of brownish-black pigment here shown look very similar to the lacunæ and canaliculi of bone when seen under the microscope. They may be few in number, and scattered about the periphery ; but more usually they are numerous, of moderate size, and their arrangement corresponds more or less to the direction of the smaller retinal arteries ; they often partially cover some of the vessels. In the later stages of the affection the pigmentary deposits approach nearer to the central portions of the fundus ; they also become larger, and are more isolated.

In the early stages the ophthalmoscope reveals no change in the optic disc and yellow-spot regions, nor are the blood-vessels perceptibly altered ; but as the disease advances, the disc becomes gradually pale, and finally assumes a yellowish waxy appearance ; the blood-vessels also undergo gradual diminution in calibre, and are finally reduced to mere threads, or become altogether invisible. In this last stage the pigmentary layer of the retina often disappears altogether, by which the vessels and intervascular spaces of the choroid are rendered plainly visible. Fig. 2, on the opposite page, represents an advanced case of this kind, in which there are yellow waxy pallor of the disc with absence of the lamina cribrosa, reduction of the retinal vessels to mere threads, and total disappearance of the pigment-layer of the retina. Frequently the arteries are accompanied by white lines. The stroma of the choroid is visible in the form of yellowish wavy streaks, and the large masses of pigment are plainly seen. Posterior polar cataract and opacities of the vitreous are frequently present in the later stages.

2. *Night-blindness* constitutes a marked and very early symptom of retinitis pigmentosa. Visual acuity is usually good in *bright daylight* ; but directly the sun sets, or if the patient is placed in a dimly lighted room, he is more or less completely deprived of the power of vision.

3. *Contraction of the visual field* is always present, and becomes very marked ; it consists in concentric limitation of the fields for white and for colours around the central region. This contraction also bears a definite relation to the intensity

of the illumination employed in the use of the perimeter ; the feebler the illumination the more contracted does the field become. Fig. 61 represents a tracing taken from a case of moderately advanced retinitis pigmentosa in bright daylight. The central vision was fairly good ($V = \frac{6}{18}$), but the patient could only distinguish objects situated close to the visual axis. Occasionally the central vision is affected before the peripheral. At other times, small seeing areas can be found in the peripheral scotoma.

These functional derangements—night-blindness and contraction of the visual field—are extremely distressing. From the earliest date of the disease it becomes more and more



FIG. 61.—Visual Field in Retinitis Pigmentosa (right eye).

difficult for the patients to see their way about after dark, or even in the twilight ; and with the advance of contraction of the visual field, there is proportionate difficulty in indirect vision. The patient can then see only the object directly looked at ; his freedom of movement is consequently much impaired, because he is compelled constantly to turn his head or his eyes in different directions in order to acquaint himself with surrounding objects. After a time central vision, even with good light, becomes affected, and in the end total blindness often ensues.

The symptoms usually begin in early life, while in a few cases no trouble is noticed until the age of fifteen or twenty

years. The consummation of the disease generally comes after the age of twenty or thirty years. Both eyes are similarly and simultaneously attacked. The disease is often congenital, and frequently accompanied by other congenital defects, both of the eye and other organs. Deaf mutes appear to be particularly liable to have retinitis pigmentosa.

Etiology and pathology.—The disease is essentially a primary degeneration of the retina; there is no ophthalmoscopic or microscopic evidence whatever of inflammation. The affected portions of the retina show complete atrophy of the nerve-elements (rods, cones, and fibres). There is interstitial development of connective tissue. The walls of the vessels are found to have undergone hyaline thickening, by which their lumen is greatly diminished; the finer arterioles of the periphery being completely transformed into tracts of connective tissue. The pigment is found in granular masses in the inner layers of the retina, often arranged around the vessels in their surrounding lymph sheaths; it is produced by a proliferation of the cells of the pigment-layer of the retina. Colloidal degeneration of the retinal epithelium usually takes place, and these swollen colloidal cells grow into the substance of the retina and may be found in the perivascular lymph spaces with masses of pigment. The choroid appears to be unaffected in true pigmentary retinitis, although it often presents lesions in syphilitic choroido-retinitis.

The *causes* are unknown. *Heredity* has a great influence. It is frequently found in several members of the same family, and has been traced through several generations. *Consanguinity in the parents* has been proved to exist in about 25 per cent. of the cases (Leber, Hutchinson), and congenital syphilis has been put forward as a cause (Galezowski), but is not generally accepted. Macnamara, however, states that retinitis pigmentosa is frequently met with among the Hindoos, where consanguinity in marriage is forbidden.

The *diagnosis* is easy in ordinary cases. Difficulty sometimes arises in cases of advanced syphilitic choroido-retinitis, where there is much pigment. In true retinitis pigmentosa there should be *no patches of choroidal atrophy*, and *no vitreous opacities*.

Prognosis is unfavourable, but the rate of progress is usually slow, and the patient may go on to the age of fifty or sixty before he is absolutely blind.

Treatment is unavailing. A few cases have been somewhat improved in visual acuity and in visual field by galvanism.¹ Beyond this a tonic regimen, neutral-tinted glasses, and a proper care of what sight remains, are the only means in our power.

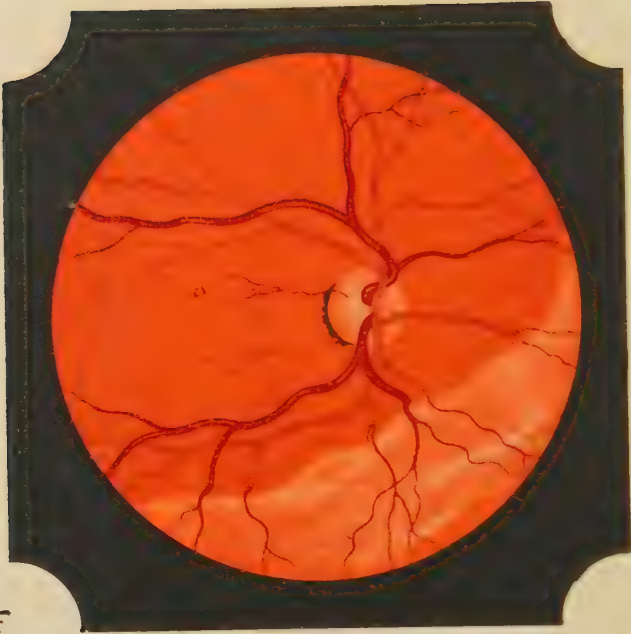
Retinitis pigmentosa without the characteristic pigment. Several cases have been recorded in which all the classic symptoms of retinitis pigmentosa were present, but where ophthalmoscopic examination failed to discover the characteristic signs. Minute dark dots were seen in the periphery of the fundus, and it is supposed that this condition is an early stage of the characteristic disease, and that the pigment has not passed inwards beyond the external granular layer.

Retinitis Punctata Albescens.—This, probably a primary degeneration of the retina, resembles in certain respects retinitis pigmentosa. The patients complain of night-blindness, but there is no concentric constriction of the visual field. The fundus differs from that seen in the pigmentary degeneration in that pigment is completely absent, but a large number of whitish-yellow spots are dotted over it, and are especially numerous in the macular region, though this part of the fundus is occasionally free. As in retinitis pigmentosa, the disc has a waxy appearance, and the vessels are constricted. The condition is usually stationary, though it may be slowly progressive. There is almost always a family history of consanguinity, and several members of the same family are usually affected.

Atrophia gyrata choroideæ et retinae of Fuchs is a rare form of degeneration of the retina and choroid, occasionally found in children. At first, small round white spots appear with healthy retina and choroid between them. Later, however, these spots increase in size and become confluent; and finally large patches of atrophy encroach on the macula and the disc. There is usually a family history of consanguinity.

¹ Gunn, *Ophth. Hosp. Reports*, vol. x. p. 161.

Plate XIII.



W.J.R.

Fig. 1.—Slight Detachment of Retina.



Fig. 2.—Severe Detachment of Retina.

DETACHMENT OF THE RETINA.

Symptoms.—By direct ophthalmoscopic examination various appearances are presented, depending on whether the detachment is due to fluid or solid growth, on the nature of the fluid or growth, and on the length of time the detachment has existed. The detachment may be slight or extensive, it may involve the whole or a part of the retina, it may occur at any part of the fundus, but if due to subretinal fluid, it is usually situated near the equator at the lower part. Whenever the retina is separated from the choroid, that part of the fundus is changed in appearance. When the detachment is recent and the retina retains its transparency, the alteration in focus, the dark colour and the wavy outline of the vessels, are the only signs. When the detachment has existed for some time, the normal orange-red aspect of the corresponding part of the fundus is generally found to assume a greyish semi-transparent or opaque appearance. When the subretinal effusion is slight and the retina transparent, there is still a red reflex from the choroid. When the detached portion of the retina is opaque, this reflex is altogether absent. When the detachment has existed for a considerable period, it is usually found to float up and down in the vitreous with quick movement of the globe. When a considerable portion of the retina is separated, its surface is found to present an undulating, rippled appearance (see figs. 1 and 2 on the opposite page). The line of demarcation from the rest of the fundus is usually distinct. The retinal vessels are seen to follow the undulations; they have the appearance of copper wire, are diminished in size, dark red or black in colour, and the arteries are without their central light streak, all these effects being optical. At the posterior edge of the detachment the vessels suddenly dip and disappear. In thus examining the detached portion of the retina by the direct method it must be remembered that whilst this is in focus, and can be best seen by a strong convex lens, the rest of the fundus is out of focus, and may require even a concave lens in order to be properly examined. By the indirect method the greyish or bluish-grey aspect of the detachment is less apparent than by the direct; and

unless the media are very clear and the detachment sharply limited, it becomes difficult to ascertain the extent of the lesion by this method. In all cases the pupil should be dilated by atropine or homatropine. Sometimes the detachment extends as far as the edge of the disc, so that a part of the latter is obscured, whilst the remainder can be seen. Occasionally the detachment extends to the whole retina, which is then pushed forwards in a funnel-shaped manner, so that it may sometimes be seen by oblique focal illumination; all fundus reflex is destroyed, and vision absolutely lost. Vitreous opacities are frequently found, and are best seen by the plane mirror; they often obscure the existing detachment.

The above are the ophthalmoscopic signs of a simple retinal detachment. If an intra-ocular growth behind, or in the deeper layers of, the retina be the cause, the appearance may be different. A small growth usually has the retina adherent to it, and if this be the case, alteration in level may be the only sign of a separation of the retina. There is no rippling of the surface, no movement; and the vessels may be normal. If the growth be pigmented, the retina over it will appear dark, and the vessels of the neoplasm may be seen through the transparent retina. Hæmorrhages may be seen in the detached portion.

The *functional troubles* of this lesion are severe and characteristic. The onset is usually sudden, but only one eye may be affected, so that the patient is not always aware of the change, and may not discover it until some time afterwards. Generally, however, the patient notices a sort of cloud appear before the eye, which obscures the sight. This at first is frequently central, total blindness resulting; but after a day or two the sight considerably improves, though the upper part of objects looked at is hidden. This means that the central part of the retina, at first detached, falls back to its normal position, owing to the gravitation downwards of the sub-retinal fluid, which now produces a more peripheral detachment. The visual field is found to present a scotoma corresponding to the detached portion of the retina. A careful examination in this direction should be made, inasmuch as the scotoma often extends over a greater area than the corresponding

apparent detachment; we may thus learn that the adjacent parts are threatened with further separation, which indeed has already commenced. Objects sometimes appear to be distorted in different ways (metamorphopsia).

The *tension* of the eyeball may be normal, raised, or diminished. In traumatic detachments of the concussion variety it usually remains normal; in myopic detachments there is generally, and in penetrating injuries always, diminished tension; if the detachment is caused by an intra-ocular growth the tension is usually, if by hæmorrhagic glaucoma always, raised.

Premonitory symptoms, as *muscæ volitantes*, are sometimes observed; patients also complain of subjective sensations of flashes of light, and perhaps coloured vision, erythropsia being the commonest.

Etiology and pathology.—The causes of detachment are various and may be divided into the following: (*a*) diminished pressure in front of the retina; (*b*) increased pressure behind the retina; and (*c*) traction.

Owing to the very incomplete attachment of the retina to the choroid, it is readily understood that any diminution in the pressure in front of the retina, or any increase in the pressure behind it, will cause a separation of the two membranes. One of the commonest conditions in which detachment is found is that of high myopia, the probable reason being that the retina is stretched between its anterior and posterior attachments during the enlargement of the eyeball, and finally gives way, the liquefied vitreous passing through the rent into the post-retinal space, and bulging the retina forwards. In favour of this view is the fact that a rupture in the retina is often found in such cases, either ophthalmoscopically or after enucleation. These ruptures are often circular in shape, and probably produced differently: a patch of chorio-retinitis results in a localised adhesion between these membranes, and, as a consequence, the retina here remains in contact with the choroid, being separated from the remaining detached portion. Other causes of diminished pressure in front of the retina are perforating wounds of the eyeball and perforating ulcers of the cornea. With these must be classed

that form of detachment which, unfortunately, occasionally follows the operation of cataract extraction. If the vitreous be unusually fluid, if the patient squeeze during the operation, if the incision be made too peripherally, if undue pressure be made during the delivery of the lens, vitreous may present in the wound and escape, retinal detachment, more or less extensive, being a frequent ultimate result.

Again, disease of the vitreous itself, leading to a fall in tension, may produce detachment probably in one of several ways. In synchysis, where the vitreous is very fluid and the tension low, detachment may occur. Subsequent to suppurative hyalitis, where the puro-lymph is becoming organised or inspissated, forming the so-called pseudo-glioma, bands of tissue exert a traction action on the retina, to which they are attached as a result of inflammatory processes, and drawing it forwards, produce its detachment. Nordenson believes that a fibrillary condition of the vitreous, the result of a primary degeneration and not necessarily of previous inflammation, is often the chief agent in the production of retinal detachment.

Pressure behind the retina may be due either to a solid growth or to a collection of fluid. The solid growths are, choroidal sarcoma, which is the commonest, choroidal secondary carcinoma, and the exophytum variety of glioma retinae. Fluid often accompanies these growths. Fluid alone, however, may be present, constituting the so-called serous detachment. A mechanical exudation from the choroidal vessels may result from diminished intra-ocular pressure; or an acute choroiditis may cause an inflammatory exudation; or injury may produce a subretinal hæmorrhage.

The post-retinal fluid is albuminous; it usually contains lymph, fat, blood, epithelial cells, and pigment. The vitreous is often more fluid than normal, usually contains opacities, and frequently shows a fibrillary degeneration. After prolonged separation, the structure of the retina presents atrophic changes.

A clinical classification of the etiology of retinal detachment may be made as follows: (a) traumatic, including concussion of the eyeball, perforating injuries, and perforating corneal ulcers; (b) progressive myopia; (c) inflammatory causes, such as irido-choroiditis and panophthalmitis; and (d) new-

growths. Besides these varieties, there is a fifth class : (e) the idiopathic detachments, for which no adequate cause can be discovered.

Retinal detachment occasionally occurs in chronic Bright's disease.

Diagnosis.—Most cases of retinal detachment are diagnosed readily. In many the history is extremely suggestive. 'A cloud gradually falling over the sight from above' is the result usually of detachment. Sudden blindness in a highly myopic eye means total detachment.

The ophthalmoscopic signs are pathognomonic, but in early cases the condition may be missed if the peripheral parts of the fundus are not carefully examined. In some cases it may be impossible to see the fundus details, owing either to vitreous opacities or to opaque lens, and here recourse must be had to other methods. It may be stated as a rule, almost without exception, that vitreous opacities in a highly myopic eye denote retinal detachment. The retina of an eye with an opaque lens may be tested by requiring the patient to localise a beam of light thrown into the eye so as to fall on the various parts of the fundus.

But it is not so much the diagnosis of retinal detachment itself, as the diagnosis of its cause, that is important. A full discussion of this subject will be found under Sarcoma of the Choroid (p. 222).

The *prognosis* is generally unfavourable. Even in the best cases, where the disease remains stationary, the vision is always defective, and we are never certain that the affection may not extend to the rest of the retina. A few cases of spontaneous recovery are on record, and some good has been effected by treatment. It must be borne in mind that where one eye only is affected in myopia, the second eye is generally in danger of a similar attack.

Treatment.—Retinal detachment due to choroidal sarcoma or retinal glioma demands immediate radical operation; the eyeball must be enucleated, together with a considerable part of the optic nerve. When there is any doubt as to the presence of a growth, the post-retinal space should be aspirated, in order that the nature of the detachment may be ascertained; while

absence of fluid indicates the existence of a growth, its presence does not exclude one. The combination of retinal detachment with glaucomatous symptoms and loss of vision is an almost invariable indication for enucleation.

Retinal detachment due to secondary carcinoma should not be treated by enucleation until glaucomatous symptoms arise.

Retinal detachment due to the presence of fluid alone in the post-retinal space has been treated in various ways, many operations having been devised to get rid of this fluid, and so bring the retina back to its normal position. Sichel, in 1850, suggested puncture of the sclerotic, but he resorted to it only in the last stage of the condition to give relief from inflammation. Von Graefe and Bowman were the first to practise to any great extent tapping of the post-retinal space. This was done from within, so that the retina was divided and the fluid was able to enter the vitreous. Von Graefe laid stress on the necessity of dividing freely any membranous opacities in the vitreous humour. Deutschmann advocated the method of Von Graefe, but divided still more freely any adhesions between vitreous and retina. De Wecker, after making a post-retinal puncture, inserted a wire drain, and so kept the external orifice open.

Various methods to increase the pressure in front of the detached retina have been suggested and resorted to. Samelsohn attempted to increase the intra-ocular tension by means of prolonged external pressure. Karl Grossman drew off the subretinal fluid, and then injected a sterilised normal saline solution into the vitreous. Deutschmann tried the injection of the vitreous humour of a rabbit. H. Schoeler advocated the injection of a glycerine solution of iodine into the pre-retinal space. This method was modified by Badnel, who first removed the post-retinal fluid. Recently, electrolysis has been used to some considerable extent by Terson and others.

Most of these operations have in a few instances been attended by partial replacement of the retina, slight improvement in visual acuity, and diminution in the size of the scotoma; but these benefits have rarely been permanent.

The operation most commonly performed is simple

puncture or aspiration. Having ascertained by ophthalmoscopic examination the exact position of the detachment, the eyelids are separated by a speculum, and the globe is held firmly by the fixation forceps in such a position that the detached portion is brought towards the front; a broad needle or a Graefe's cataract-knife is then plunged through the conjunctiva and the tunics of the globe into the middle of the detachment; in doing this the point of the instrument should be directed towards the centre of the globe—that is, away from the lens. In the act of *slowly* withdrawing the instrument, its blade may be half rotated whilst between the lips of the wound; this will facilitate the escape of the subretinal fluid. After the operation a light compress is applied and the patient kept quietly in bed. This method has in a few instances been attended by partial replacement of the retina, considerable improvement in visual acuity, and diminution of the visual scotoma. In the majority of cases, however, it has been of no perceptible benefit, and in a few the eye has become much worse after the puncture.

The simplest form of treatment, and one which has probably had as many successful results as any of the operations that have been suggested, is that of rest. This is especially indicated in recent traumatic cases, though some cases of myopic detachment cured by rest are on record. With the rest in bed should be combined rest to the eyes, best procured by atropinisation and protection from light by a shade or smoked glasses. The diet should be low, and various general measures to produce depletion may be added. Of these, pilocarpine has been warmly advocated. It is best administered as the hydrochlorate in the form of hypodermic injections, and its action is assisted by giving by the mouth saline purges, iodides, or salicylate of sodium. Other auxiliary measures are the application of leeches over the mastoid process or temple, hot-air baths, and a pressure bandage.

Prophylactic measures would appear to be most strongly indicated in this affection. In the case of high myopia, for example, it is of the greatest importance that the error of refraction should be corrected by the use of proper spectacles, whilst the eyes are spared from all strain and, indeed, as

much as possible from ordinary work. The prophylactic treatment of retinal detachment is, indeed, the treatment of progressive myopia, to the account of which the reader is referred.

TUMOURS OF THE OPTIC NERVE AND RETINA.

Primary Tumour of the Optic Nerve.—Though this is a rare disease, still many instances have been recorded.

Etiology and pathology.—This condition frequently appears to be congenital, and a large proportion of the cases have been in children under fifteen years of age. The tumour is rarely very malignant, being generally encapsuled and of slow growth. It spreads, as a rule, inwards towards the cranial cavity, and only very exceptionally does it involve the eyeball. Many varieties are on record—namely, fibromata, sarcomata, gliomata, myxomata, neuromata, endotheliomata, &c. Generally, however, the growth combines the appearances of two or more varieties; thus, glio-sarcomata, fibro-sarcomata, and fibro-myxo-glio-sarcomata have been described.

Symptoms.—Early blindness is followed by gradually increasing proptosis. There is rarely any lateral displacement, and the movements of the globe long remain unaffected. On attempting to push the globe backwards, resistance is felt. The fundus often shows optic neuritis or atrophy, and the retinal veins are usually engorged. As the growth increases, lagophthalmos results, leading to corneal haze and ulceration. Should the tumour be near the globe, increasing hypermetropia may be noticed. As a rule, however, the starting-point of the neoplasm is at the entrance of the retinal artery into the optic nerve, *i.e.* 12 mm. behind the globe.

Diagnosis.—The diagnosis is often difficult. The characteristic signs are the early blindness, followed by protrusion of the globe directly forwards, with little impairment of motility, and no evidence of inflammation.

Prognosis.—If the tumour can be completely removed, the prognosis, as far as the patient's life is concerned, is good. Otherwise, recurrence is almost inevitable. Metastasis is rare. If death occurs, it is on account of involvement of the brain, the tumour spreading by direct continuity.

Treatment.—In nearly every case, the orbit should be completely emptied. Some instances in which the globe has been left have been recorded.

Secondary tumours of the optic nerve are even more rare than the primary variety. They are usually secondary to intra-ocular tumours. A few cases of metastatic growths of the optic nerve have been recorded.

Tumours of the optic-nerve sheath resemble tumours of the nerve itself. The proptosis is usually somewhat lateral, and the mobility of the globe is often affected. Vision is lost later, and consequently diplopia is a common symptom.

Primary Tumours of the Retina.—A few cases of primary sarcoma of the retina have been recorded. The only tumour, however, of importance is glioma retinae.

Glioma of the Retina.—*Symptoms.*—*In the early stage* the ophthalmoscope reveals one or more brilliant white or pinkish-white patches in some part of the retina, which in glioma exophytum is detached. These patches differ considerably from those of retinitis in being of a brighter, more metallic, lustre; they are sometimes vascular, and hæmorrhages occasionally exist. The tension is normal or slightly diminished. There are no external changes in the appearance of the eye, no pain is complained of; the eye is quite blind, but this is not discovered owing to the youth of the patient; hence the disease is rarely seen at this early period; it usually passes unnoticed until the growth has become sufficiently large to be visible through the pupil; it is then detected by the parents, and sooner or later the patient is brought for advice. In this, *the second stage*, the pupil of the affected eye, as a rule, becomes considerably dilated, and the anterior chamber shallow. The tension usually remains normal, owing to the elastic condition of the ocular coats; it may, however, be somewhat raised. The pupil no longer has its normal black colour, but presents a white, pink, or yellowish lustrous look, the 'cat's eye' appearance. *By focal illumination* the tumour may be observed to project into the vitreous cavity; the surface may be smooth or nodulated; and some blood-vessels can generally be seen upon the white background, and perhaps a few hæmorrhages. *By the ophthalmoscope* a similar condition

is observed. The lens and vitreous are usually clear. In this stage there is often pain in the eye, and inflammatory symptoms are liable to supervene in the form of congestion of the scleral vessels. As the growth increases the lens is pushed forward and may become opaque, the anterior chamber becomes more shallow, the iris and ciliary body atrophy, the cornea becomes dull and opaque, and loses its sensitiveness; the eye, in fact, becomes glaucomatous. The onset of glaucoma may be sudden, but generally is insidious. As the growth continues to increase in volume, the tunics of the globe can no longer sustain the intra-ocular pressure, and usually become ruptured in the region of the sclero-corneal junction. In this, *the third stage*, the tension is suddenly decreased, and the disease rapidly extends to the surrounding parts, and backwards along the course of the optic nerve to the brain. Occasionally an enormous growth takes place forwards, the protruding ulcerating mass being the size of a foetal head. In *the fourth stage*, metastasis occurs.

Etiology and pathology.—Glioma almost always occurs in early life, either intra-uterine or during the first three or four years; occasional cases have been recorded up to thirteen years. Several members of a family have been known to be affected. No relationship between glioma and injury has been proved to exist. When an eye with glioma is opened during the second stage the tumour presents a yellowish-white appearance; it contains blood-vessels, hæmorrhages are seen, and in some parts there are calcareous particles. The hinder part of the retina is the most frequent site. *Histologically* this new-growth largely consists of small round cells similar to those found in the granular layers of the normal retina, but many of them have short projections; they are embedded in a small amount of intercellular substance. Each cell is a rounded body about 8μ in diameter, and contains a large freely staining nucleus, in the centre of which are a few granules. Blood-vessels are found in the tumour, but are not especially numerous; they are not in actual contact with the cells, but usually have a distinct sheath, probably a lymph space; outside this clear space is found a zone of cells which stain freely; beyond these the staining becomes more feeble,

and the cells are found to have undergone either fatty, hyaline, or calcareous degeneration. Finally, the vessels become destroyed, and the whole glioma degenerates from absence of blood-supply. In many cases the cells are arranged as many-layered tubes surrounding a blood-vessel. If we examine the free or spreading edge of the tumour, we find that the granular layers and the layer of nerve-cells are the parts first attacked. The origin of these cells has been much disputed. Recently, by the staining methods of Golgi and Ramón y Cayal, many of them have been shown to be quite similar to neuroglial cells. Other cells, those with the single process, appear to be ganglionic cells, while sometimes long cylindrical cells have been seen arranged in groups surrounding a single lumen; these cells appear to be derived from the constituents, the rods and cones, of the external layers of the retina. Two chief kinds of glioma are recognised, viz. *G. exophytum* and *G. endophytum*. *Glioma exophytum* commences in the inner granular layer, which becomes thickened to join the outer; a diffuse thickening of the whole retina is formed, with nebular bulging on its outer side, from which the disease extends to the choroid. This is the form most frequently met with; and as it does not perforate the internal limiting membrane until a late stage, the smooth contour of the growth is maintained, which accounts for its glistening nature. *Glioma endophytum* commences in the granular and nerve-fibre layers of the retina. The parts of the retina which are not *at first* attacked would seem to be the rods and cones, the molecular layers, the system of Müller's fibres, the basement or limiting membranes, and the pars ciliaris retinæ. The structures which are attacked appear to be the nerve-elements and the very delicate neuroglia. *The mode of extension of glioma is important. Its chief direction is along the fibres of the optic nerve.* Here the cells first plug up the optic disc so as to push back the lamina cribrosa; after a time they appear on the outside of the latter, in the form of clusters occupying the bundles of nerve-fibres; the coarse trabecular tissue is but little affected, even in advanced cases. The central artery and vein are not attacked. From the optic nerve the cells sometimes get into the nerve-sheath, and thence extend to the intracranial meninges,

occasionally also to the diploë of the cranial bones. Sometimes, in about 20 per cent. of cases, the child has glioma in the other eye, but we have no evidence to prove that the disease spreads from one eye to the other; in fact, pathological examination has shown that this does not take place. Where both eyes become affected, we have an instance of two primary growths. There may be a considerable interval between the appearance of the two growths.

Another mode of extension is by way of the choroid; glioma exophytum usually spreads in this way. The part of the choroid first invaded is that nearest to the optic disc. When the cells get into the choroid itself they immediately increase by multiplication, and the tissue of the choroid is destroyed, its place being occupied by a thicker layer of glioma cells. These cells then extend to the sclerotic, which is attacked in the direction of its component fibres. They then pass forwards along the suprachoroidal lymph space, through the fibres of the ligamentum pectinatum, into the anterior chamber. They may thus push back the lens; sometimes they cause necrosis of the cornea. When not in the anterior chamber they may cause forward bulging of the lens. The vitreous undergoes atrophy, and causes a peculiar wavy appearance of the retina (detachment).

Metastasis sometimes, though rarely, takes place. The commonest places for the secondary growths are the brain, the cranial bones, and the lymphatic glands. Metastatic growths have also been described as having been found in the parotid, the skeletal bones, and the liver. Metastasis usually takes place by means of the blood-vessels. *Heredity* appears to play an important rôle in the existence of these tumours; two or more children of the same parents may suffer, and a history of cancer of the eye during the early part of the parent's life may sometimes be elicited.

There is still considerable difference of opinion as to whether gliomata should be classed under the sarcomata or the epitheliomata, though at the present time the evidence is rather in favour of the latter. There are many important differences between an intra-ocular glioma and an intra-ocular sarcoma as found in adult life. The cells, though similar in many respects,

have important differences; spindle cells are absent from gliomata, and neuroglia cells, as shown by special methods of staining, are numerous. Gliomata frequently occur bilaterally as two primary growths, whereas sarcomata are never found primarily in both eyes. Secondary sarcomatous growths are usually found in the liver, which is not the case in gliomata. Gliomata are very malignant, but rarely, if ever, recur after three years; sarcomata, though less malignant, may recur after a considerably greater interval. If a glioma is a form of sarcoma, it must originate from the retinal blood-vessels, since this is the only tissue that is mesoblastic in origin, the nervous elements and the peculiar connecting tissue (neuroglia) being derived from epiblast. No observer has, however, recorded the fact that he has seen a specimen of glioma starting from a blood-vessel.

Diagnosis is usually easy. Given a lustrous white or pinkish-white tumour, occurring in a young child, where there have been no perceptible inflammatory symptoms, and where the intra-ocular tension is increased, we have little hesitation in pronouncing this to be glioma. There are, however, certain rare conditions¹ which closely simulate glioma, and which must be excluded before we arrive at a diagnosis.

1. From subacute and chronic suppurative choroiditis (see p. 207) found in children as a result of an acute specific fever, basal meningitis following middle-ear disease, the entrance of a small foreign body into the vitreous chamber, or from some other cause. The clinical features of this condition are such that a diagnosis from glioma is usually not difficult. The reflex from the surface of the inflammatory exudation, which is, as a rule, usually flush with the back of the lens, is not brilliant or *glistening*, it is yellowish-white in colour, whereas glioma is white or pinkish-white, and the tension is generally *reduced*. Another important difference consists in the appearance of the iris; in glioma the whole of this is pushed forwards towards the cornea, but in these forms of choroiditis the contraction of the inflammatory

¹ The conditions have been included under the term *pseudo-glioma*, which is as vague as it is unscientific, and for these reasons its use should be discontinued.

products in the vitreous causes retraction of the ciliary edge of the iris, so that the latter presents the appearance of a truncated cone with its narrow end projecting forwards. Other points, such as evidences of old iritis, a history of fits or convulsions with retraction of the head, and presence of otitis media, assist in the diagnosis. Hill Griffith¹ has pointed out that ophthalmitis may complicate a glioma of the retina, the former being visible and the latter not; the term *crypto-glioma* has been given to this condition. An exudative irido-cyclitis is set up, with a resulting exudation into the anterior part of the vitreous chamber. Subsequently phthisis bulbi may take place. He is of opinion that the presence of a pseudo-glioma in a child's eye accompanied with increase of tension demands early enucleation.

2. From a congenital membranous condition of the vitreous fluid (see p. 419). Apart from the history of the case, a diagnosis between this developmental abnormality and glioma retinae is almost impossible.

3. From a tuberculous tumour of the choroid (see p. 210). In this disease the primary seat of infection can usually be found, and consequently glioma excluded. Glioma is often, tuberculous disease of the choroid never, bilateral.

4. From sarcoma of the choroid. This, however, is very rare in children, and since the treatment is the same, the differential diagnosis—which in early cases can only be made with the help of the microscope—is unimportant. In advanced cases, where secondary growths have appeared, if the abdominal viscera are affected, sarcoma is more probable than glioma.

5. Other very rare conditions, which somewhat simulate glioma retinae, are retinitis proliferans and subretinal cysticercus.

Prognosis.—Recurrence is the rule; cure the exception. The child may be considered cured if it survives enucleation for three years. Should death occur, cerebral symptoms are usually present. Double glioma does not contra-indicate enucleation, and does not make the prognosis worse. The prognosis is usually favourable if the vitreous chamber is not occupied to a greater extent than one-third at the time of enucleation, and if the cut end of the optic nerve is healthy.

¹ *Med. Chron.* April and May, 1892.

The *treatment* of glioma consists in the *immediate removal* of the whole of the affected globe and as much of its optic nerve as possible. By this means the disease is prevented from spreading backwards to the brain and in other directions, so that, although the eye is lost, the patient's life may be saved. After excision of the globe in this manner, the cut end of the optic nerve should be examined microscopically. If this is of normal size, and contains no glioma-cells, we may hope for a good result. If slightly swollen, and a few of the nuclear bodies are found, the result is doubtful. If much swollen, and numerous nuclear bodies are found, there will probably be a return of the disease in the optic nerve in the course of a few months. Implication of the optic nerve must be met by total extirpation of the orbit.

When the disease has perforated the ocular tunics and affected the surrounding parts, the whole of the contents of the orbit must be extirpated, although the prognosis is extremely grave.

In the later stages, where a large necrotic mass of tissue exists, constant irrigation with strong antiseptics is all that should be attempted.

Spontaneous cure never occurs. Should the tumour be left to itself, death takes place in from eighteen months to two years, either on account of general cachexia, or with cerebral symptoms.

Subretinal Cysticercus Cellulosæ.—The scolex of the *tænia solium*, when intra-ocular, usually first develops behind the retina (see p. 418).

CHAPTER VIII.

THE VISUAL FIELD, AND THE USE OF THE PERIMETER.

THE VISUAL FIELD—THE PERIMETER—SCOTOMATA—MCHARDY'S PERIMETER
 —PRIESTLEY SMITH'S PERIMETER—THE IMPORTANCE OF PERIMETRY—
 THE FIELD OF FIXATION—THE ANGLE 'GAMMA.'

The visual field is the extent of a plane at right angles to the visual axis, over which the eye can recognise objects. Thus the eye being fixed on any point, 'the fixation point,' the image of this point will fall on the yellow spot (direct vision); at the same time other objects will be less distinctly seen by the peripheral portions of the retina (indirect vision). The objects most distant from the fixation point will represent the limits of the visual field, and the latter may be considered as subtending a cone-shaped space whose apex is situated at the eye, and whose base becomes larger in proportion to its distance from the eye.

If the yellow spot were the only portion of the retina used for visual purposes, we should suffer the greatest inconvenience from being able to see nothing but the object to which the visual axis was directed; all side objects, such as passers-by in the street, the ground on which we walk, and the thousands of other objects which we see indirectly with the peripheral portions of the retina, could then be observed only by the constant turning of the eyes or head.

The limits of the visual field may be roughly ascertained in various ways.

1. Place the patient with his back to the window or artificial light; let him close one eye, and with the other look straight at your nose at a distance of about two feet; then hold up your two hands on opposite sides of your nose in the plane

of the face, and ascertain to what extent they can be separated in the vertical, horizontal, and oblique directions before they disappear from his indirect vision.

2. The perimeter consists of the arc or quadrant of a circle which in turning on a point describes a hemisphere, at the centre of which the eye of the patient is placed. The hemisphere thus described must be amply illuminated by diffused white light. At the pole of the hemisphere which is opposite to the patient's eye is a white spot, which the patient can fix by direct vision. The arc is divided into degrees, starting from 0° , which marks the white spot, up to 90° . These divisions are marked upon the arc. The test-objects should consist of small discs of white and coloured paper, of 3 to 10 mm. diameter. In order to ascertain the limits of the visual field we proceed as follows:

The head being fixed, and the eye to be examined being placed at the centre of the hemisphere, the other eye is covered with a shade. The patient is then told to look steadily at the white spot above mentioned, while the surgeon, placing himself behind the perimeter, keeps a watch on the patient's eye, so as to be able to check its least deviation from the centre. Then, the arc of the perimeter being held in a certain plane, the vertical for example, the test-object is advanced from the periphery towards the centre of the arc until it is just recognised by the eye under examination. This point corresponds to the limit of the visual field for that meridian.

The horizontal and oblique meridians are then similarly ascertained, and the data are transcribed on to a diagram or 'chart,' such as is shown in fig. 62, which represents the projection of a hemisphere upon a plane surface. In that diagram we have a series of concentric circles cut by numerous radii or diameters. The centre corresponds to 0° or the point of fixation, and the diameters to the different planes in which the measurements have been made. At the extremity of each radius a number shows the inclination of the corresponding meridian to the vertical. The radii themselves are also divided into equal parts, each corresponding to 10° of the divisions upon the arc of the perimeter.

Thus, supposing the right eye to be under examination for

white, and we find the limits of indirect vision in the horizontal meridian to extend to 90° on the outer side, 70° on the inner; we proceed to mark these by dots or pricks upon the horizontal line of the chart at the points corresponding to 90° and 70° on the outer and inner parts respectively. The other meridians are similarly measured and marked off; and the dots are finally joined together by a continuous line, in the manner represented in fig. 62.

In this way we find that the normal visual field is not circular, but that it is more extensive in the outer and lower than in

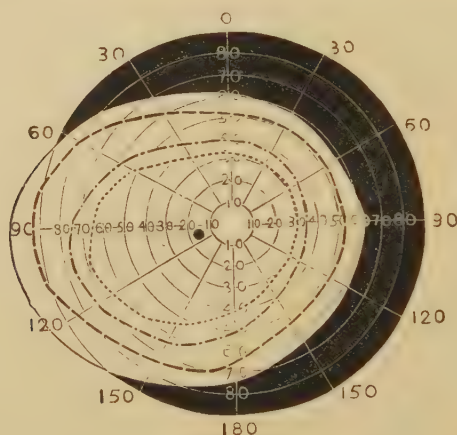


FIG. 62.—Normal Visual Field (left eye).

— — — blue, — — — — red, green.

the inner and upper portions. This is due partly to the fact that the retina extends slightly farther forwards on the inner side (which of course corresponds to the outer side of the visual field), but chiefly to the circumstance that the outer part is less used than the inner, in consequence of the projection of the nose shutting off peripheral rays coming from the inner side.

Visual field for colours.—In testing the limits of the field for colours, Landolt¹ found that when colours of great intensity, such as those of the solar spectrum, are used, they can be recognised quite up to the limits of the field for white. When,

¹ *Examination of the Eyes*, 1879. By E. Landolt.

however, ordinary discs of coloured paper of about 2 cm. diameter are used in the manner above indicated for white, it is found that the peripheral portions of the retina are less easily excited by coloured than by white discs, and that each colour has its own limits, beyond which it ceases to be perceived by the retina. Thus, if we test the normal eye with the fundamental colours, blue, red, and green, in ordinary bright, well-diffused daylight, we find that on passing the test-object from the periphery towards the centre, the blue is the first to be recognised, after that the red, and next the green. If the fields for each of these colours are respectively taken, and their outlines marked with coloured pencils, we obtain a chart similar to that represented in fig. 62. Thus we find that the field for blue is almost as large as that for white, that it is larger than that for red, whilst the field for green is considerably smaller than that for red.

Of the other colours of the spectrum, the field for yellow is very similar to that for blue; the field for orange exists between the limits of the yellow and the red. Violet is a difficult colour to test; it appears for a considerable distance as blue before its colour is really recognised as violet. The three fundamental colours, red, green, and blue, are all that are required in practice.

The limits of the normal fields for colour are necessarily difficult to fix with accuracy, because, as we have seen, the sensibility of the retina varies with the intensity of the colour and the brightness of the illumination; and the acuteness of vision for colour is much less marked at the periphery than towards the centre of the field. The normal field of vision also depends upon the size of the object, the attention and intelligence of the patient, and the refraction of the eye, being greater in hypermetropia than in myopia.

From a number of experiments on this subject, Landolt is of opinion that the following should represent the minimum extent of the normal visual field for colours :

			Blue	Red	Green
Upper	.	.	50°	35°	30°
Outer	.	.	80°	70°	55°
Lower	.	.	55°	45°	35°
Inner	.	.	50°	40°	30°

Other observers, however, consider that the normal limits are considerably less than this. This difference of opinion is doubtless owing to different conditions in experimenting.

Scotomata.—Having ascertained the limits of the visual field, it is necessary to examine its area in order to ascertain if there exist any blind spots (scotomata). Scotomata may be *relative* or *absolute*. When relative, the test-object is only obscurely seen over the area affected; or certain colours may be lost, whereas others are retained. An absolute scotoma is present when the object is completely lost to view over a limited area of the field. To find them, the test-object should be passed from the periphery of the field towards its centre in the different meridians. The patient fixes the white spot on the perimeter as before, and is instructed to give a sign the moment that the object becomes obscure or entirely disappears, and when it again becomes clearly visible. These points are then recorded on the chart. Scotomata may be *subjective* or *objective*. If complained of by the patient, they are subjective; if discovered only by the perimeter, objective.

The blind spot of Marriotte.—There is one part of the field in which a scotoma is always present; this corresponds to the optic disc, where, as we have seen, the retina does not exist. In emmetropic eyes the position of this, the blind spot, is about 15° to the outer side, and 3° below the centre of the field. In hypermetropia this distance is greater, and may be as much as 19° ; whilst in myopic eyes it is less, and seldom exceeds 11° . The form of the blind spot is usually round, and its diameter subtends an angle of 5° to 6° .

Scotomata for colours are tested for in the same way as those for white.

Numerous forms of the perimeter have been introduced since this instrument was first used by Aubert. For a long time I was in the habit of using the instrument known as Förster's perimeter; this is, however, an exceedingly cumbersome apparatus, consisting of a broad semicircle of wood, which can be rotated about its centre; the test-object is moved along this by a system of pulleys. There is no advantage in using a semicircle, and most modern instruments have a quadrant only.

The self-registering machine invented by McHardy, and that introduced by Priestley Smith, are very excellent for the rapidity, accuracy, and facility with which they can be employed.

In McHardy's perimeter as modified by Lang (fig. 63) there is a chin-rest (*a*); the height of the rest and of the stem which supports the quadrant can be regulated to suit different patients.

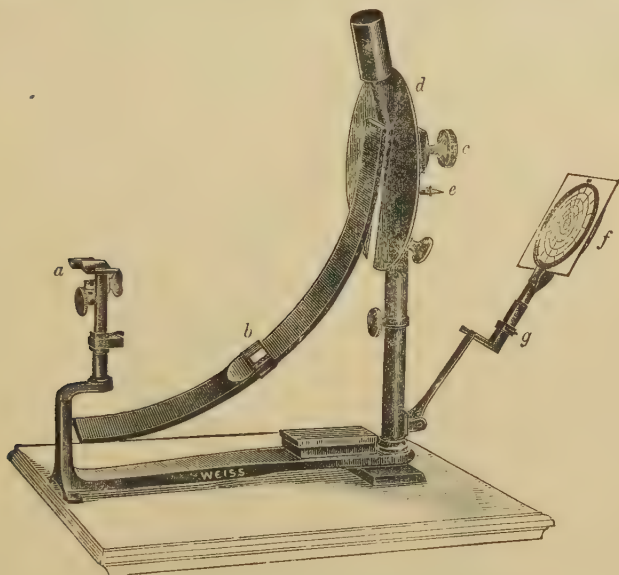


FIG. 63.—McHardy's Perimeter, modified by Lang.

The test-object—a disc of white and coloured paper—is fixed on a traveller (*b*), which is moved by an endless band worked by rotating the milled head (*c*). The hand of the surgeon can be concealed while rotating this by affixing the shield shown detached in the figure (*d*).

The chief advantage of the instrument is the mechanism by which the registering of the field is accomplished. The milled head (*c*), in addition to moving the traveller (*b*), rotates two toothed wheels, which cause the sharp pointer (*e*) to move in the same direction as the traveller, but at a diminished speed, so that when the traveller is at the fixation point (zero) its extremity lies exactly behind the same point.

The chart is placed in a frame (*f*) supported on a hinged limb (*g*), in such a position that when the traveller is at zero the pointer corresponds to the centre of the chart.

The quadrant can be rotated to, and fixed in, any position ; and as the pointer moves with it, its line of movement always corresponds with the position of the quadrant.

In using the instrument, when the traveller has reached the limit of the visual field the chart is pressed against the pointer ; the position of the test-object is thus recorded on the chart by a puncture.

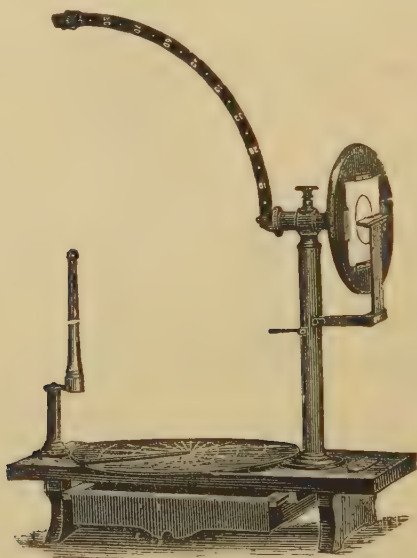


FIG. 64.—Priestley Smith's Perimeter.

Priestley Smith's Perimeter.—The general arrangement of the instrument will be understood from fig. 64 ; the following points, only, need be particularly described.

1. The patient rests his cheek against the wooden pillar, so that the eye is about an inch and a half above the knob, and vertically over it. The height of the instrument is regulated by movable blocks.

2. The quadrant, which is a flat strip of metal engraved upon its two sides, is rotated by a wooden hand-wheel attached

to the axis ; it is balanced by a weight upon the hand-wheel, so that it will stand in any position without being fixed.

3. The test-object is a square of paper gummed upon a light vulcanite wand which the operator holds in the left hand. With the right hand he rotates the hand-wheel, and pricks the chart.

4. The chart is placed upon the hinder surface of the hand-wheel, and rotates with it. There is a mark on the hand-wheel to show which way the chart is to be placed. This mark is brought to the top, and the chart is then slipped in from above downwards and in the upright position.

5. Immediately behind the hand-wheel is fixed a horizontal scale, the divisions of which correspond with the circles on the chart. As the quadrant rotates the chart rotates with it ; and in whatever position the quadrant stands, the corresponding meridian of the chart stands against the scale. This arrangement enables the operator to prick off his observations with the greatest ease, and has the further advantage that the chart is constantly under inspection, and that any portion of the field can be immediately brought under examination at any time.

6. The charts are of two kinds, A and B. The A charts correspond to the entire field, and are divided by circles from 0° to 90° , the limits of the average normal field being shown by a dotted line. The B charts are for mapping the central part of the field on a larger scale, and are divided from 0° to 45° . The scale of the perimeter is graduated accordingly on its two sides : the A side is to be used with the A charts, and the B side with the B charts.

7. There are many cases in which it is better to sweep the field, or parts of it, in circles than in meridians, *e.g.* hemiopic and sector-like defects, in which the boundary-line of the field runs in a meridional direction. In cases of this kind, the test-object may be placed in the clip which slides upon the quadrant, and carried round the field in successive circles. (*Vide* 'Ophthalmic Review,' November 1882.)

Importance of Perimetry.—The importance of systematic observation by means of the perimeter is paramount. There is scarcely a lesion of the interior of the eye which is not

accompanied by perimetric symptoms. Not only does it frequently assist in establishing a diagnosis which without its aid would have been doubtful, but a prognosis of the case can often be effected by this means which would otherwise have been impossible. Thus, in *atrophy of the optic nerve* there is always found to be a contraction of the visual field at least for colours (see p. 273); and although this affection is easily recognised in the advanced condition, yet there are many cases occurring in practice where, at the onset of the disease, the discs are not particularly pale, nor are the vessels contracted. Under such circumstances the discovery of contraction of the visual field for *colours* is of great assistance, both in the diagnosis and in the prognosis of the affection. In advancing optic atrophy, the contraction of the visual field is almost as constant a symptom as the failure of acuteness of vision.

In *glaucoma* the contraction of the visual field is usually quite characteristic of the disease (see p. 425). First the inner, and then the upper and lower portions of the field begin to contract, and this gradually extends towards the centre of the field, the central and outer parts alone remaining unaffected. At a later period even the central vision is abolished, leaving only a portion of the outer part of the field intact. Finally even this is lost. The remarkable feature of this diminution is that the contraction of the fields for colours appears to advance at the same rate as that for white, and so retains throughout a *concentric* arrangement similar to that existing in health. There is sometimes a difficulty in distinguishing between chronic glaucoma and partial atrophy of the optic nerve. The cupping of the disc may be slight, and there may be pallor in both affections; but in the case of glaucoma the fields for colour are usually limited in proportion to the contraction for white, whilst in atrophy the colour sense, more especially for green and red, may be almost abolished. Compare the charts of optic-nerve atrophy and glaucoma.

In *pigmentary retinitis* there is also contraction of the field, which is alone almost characteristic of the disease. Here we usually find concentric limitation of the field, which involves all the peripheral portions, and leaves a small circular area

around the centre in which the vision for colours is comparatively good. Fig. 61 shows the chart of a man suffering from this affection.

In *detachment of the retina*, also, the use of the perimeter is often of service. Thus, having found by the ophthalmoscope that a portion of the retina is detached, we proceed to ascertain the limits of the field for white. Finding a limited portion in, say, the upper half of the field, destroyed, we know from this that the corresponding lower half of the retina is separated from the choroid. Now, by further testing the lower half of this field for colours we may still learn something as to prognosis. If we find the limits of the fields for colour extending quite up to the edge of the detachment, this may be regarded as a favourable indication; if, on the contrary, the colour-sense is defective at some distance from the detachment, a further separation of the retina is to be feared.

In *hysteria* the visual field is often peripherally and concentrically contracted, enlarging on cutaneous stimulation. The field tends to slowly diminish under observation, a typical spiral field being sometimes obtainable. The colour fields are frequently inverted, that for red being occasionally larger even than for white.

In the various forms of *hemianopsia* (see p. 330), again, it is advisable to keep a record of the limits of the persistent part of the field both for white and for colours. It is not at all uncommon, especially in homonymous hemianopsia, to find the persistent half of the visual field in a state of perfect visual acuity, both for white and for colours. A chart of such a field is shown in fig. 67. Now, it is possible for the central cause of this affection, such as a small hæmorrhage in the corresponding hemisphere, or the pressure of a gumma upon the optic tract, to recede, and so allow the hemianopsia to disappear; and, on the other hand, the central lesion may so increase as to extend to the opposite optic tract. A lesion in the tract causing homonymous hemianopsia is not usually attended with any peripheral limitation in the retained half of the field, whereas a lesion in the internal capsule or cortex is almost invariably accompanied by peripheral contraction.

By careful perimetric observation we may, to a great extent, ascertain the condition of things going on within.

In *retro-bulbar neuritis* of the acute variety there is usually an absolute central scotoma with no peripheral contraction either for white or colours. In this disease, however, an atypical field is by no means uncommon (see p. 261). A central scotoma is also found in central choroiditis, myopic choroiditis, and in senile degeneration of the retina and choroid. It may also be present as an early symptom of tumour of the optic chiasma.

In the various forms of *toxic amblyopia*—of which those due to tobacco and alcohol (see p. 325) are the most common—the perimeter is a valuable instrument of diagnosis, for by it we are enabled to discern a central scotoma for colours, which is pathognomonic of these affections.

Other forms of scotoma are sometimes found. *Sector-shaped scotomata* may be found in optic atrophy, and in obstruction to a branch of the arteria centralis retinae. That known as the *ring scotoma* forms a band round the point of fixation, while the adjacent portion of the field is unaffected, and may easily be overlooked unless the field be very carefully tested. A ring scotoma is not uncommonly found in those cases of syphilitic choroido-retinitis which simulate pigmentary degeneration of the retina (retinitis pigmentosa). What the significance of this and other rare forms of scotoma is we are not at present in a position to state; but there can be no doubt that the perimeter will in the future be of considerable help in the differential diagnosis of many of the cases of amblyopia without ophthalmoscopic signs, the pathology of which is at present obscure.

Mention may be made of the *flickering scotoma*, or *scotoma scintillans* of Listing, found so frequently as an aura before an attack of migraine. It is often associated with a spreading hemianopsia and the appearance of zigzag lights in the periphery of the visual field. The whole phenomenon usually lasts for from fifteen to thirty minutes, and is followed by severe hemicranial pain. It is probably due to spasm of the cerebral arterioles.

The **field of fixation** is a term used to express the amount of

angular deviation from a line at right angles to the plane of the face which can be given to the eye by its muscles. This can also be measured by means of the perimeter. The patient is placed in the same position as for testing the visual field. Instead of employing discs as test-objects, we substitute a letter of the alphabet, which is of such a size that its form can only be recognised when its image falls on the yellow spot. The slide of the perimeter, with the letter attached, is now passed from the centre towards the periphery of the arc, and the patient is told to follow it with his eye, and to give a sign as soon as he can no longer distinguish its form. The angle at which the form of the letter is lost is then marked upon a chart similar to those used for the visual field. The process is repeated for the remainder of the horizontal, the vertical, and the intermediate meridians. The points thus obtained are then connected by a continuous line, which maps out the limits of the field of fixation for that eye.

From a large number of experiments made in this way upon healthy eyes, Landolt has found that the average limitations of the normal field of fixation are as follows :

Outwards	45°	Inwards	45°
Outwards and downwards	47°	Inwards and upwards	45°
Downwards	50°	Upwards	43°
Downwards and inwards	38°	Upwards and outwards	47°

It is evident that this method of testing the movements of the eye produced by the action of the ocular muscles would be of great help in recording any deficiency in the action of these muscles. Thus, supposing the external rectus to be paralysed, we should find that the limits of the field of fixation would not extend to 45°, but would be nearer to zero in proportion to the completeness of the paralysis. Similarly for all the ocular muscles.

In the chapter on Strabismus it will be found that the use of the perimeter in tracing the field of fixation is not only useful in this way, but also in distinguishing the paralytic from other forms of squint.

The *field of fixation* can also be ascertained *objectively*. To effect this a lighted taper is passed along the arc of the perimeter instead of the letter just mentioned. The positions of

the patient and the observer are the same as before. The patient is then told to direct the eye as far as possible in the direction of the periphery of the arc. Having done this, the observer passes the light along the arc until, by keeping his own eye just behind it, he is able to see its image in the centre of the patient's cornea. Since the reflected pencil of rays only

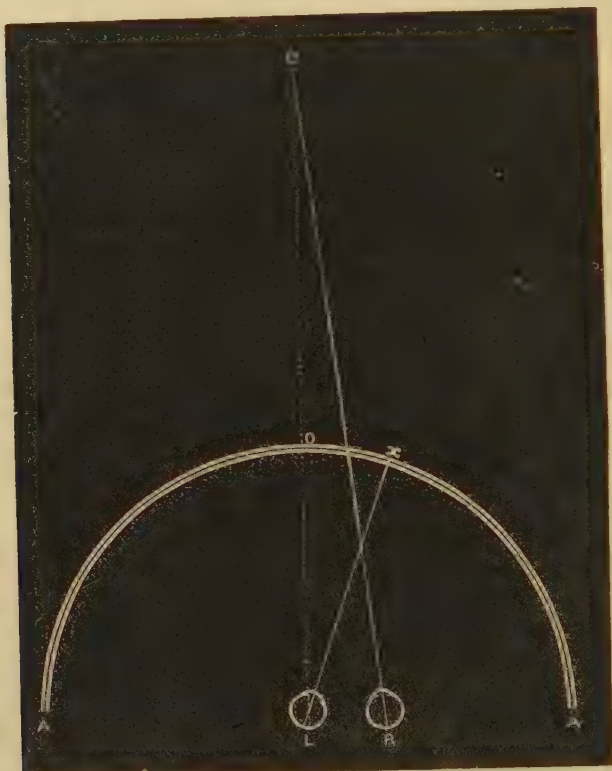


FIG. 65.—Arc of Perimeter.

coincides with the incident rays when it lies on the principal axis of the cornea, this test gives the position of the latter, and although this does not exactly coincide with the *visual* axis, the difference is unimportant. He then reads upon the arc ($\Delta \Delta$, fig. 65) the position (x) of the taper, and then proceeds to register the other meridians in a similar manner (Javal).

The *angle 'gamma,'* or the angle formed by the intersection of the line of fixation and the optic axis,¹ can also be determined by the perimeter. The patient must be made to fix the lighted taper placed at zero; the observer then moves his eye along the arc until the reflection of the flame is seen in the centre of the cornea; he then reads the angle between this point and the flame. (This experiment is accurate only when the principal axis of the cornea coincides with that of the whole eye.)

¹ See, however, p. 465.

CHAPTER IX.

AMAUROSIS, AMBLYOPIA, AND SOME FUNCTIONAL
DISORDERS OF VISION.

AMAUROSIS—TOBACCO AMBLYOPIA—HEMIANOPIA—HYSTERICAL AND CEREBRAL
HEMIANÆSTHESIA—MIND-BLINDNESS—NIGHT-BLINDNESS—SNOW-BLIND-
NESS—‘ECLIPSE’ AMBLYOPIA—MALINGERING.

Amaurosis signifies loss of sight without perceptible ocular lesions. With the advance of knowledge in ophthalmology this term is becoming less and less required, and is gradually falling into disuse. Several grades of amaurosis are recognised.

In the first grade, which is generally called *amblyopia*, there is merely diminution of visual acuity; the patient is not able to read small print with the amblyopic eye, but he can distinguish large objects, and find his way about.

In the second grade there is only *quantitative* perception of light. The patient can only distinguish light from darkness.

In the third grade, usually called *complete* or *absolute amaurosis*, both qualitative and quantitative perception of light has disappeared.

Tobacco Amblyopia (tobacco amaurosis).—This is a disease which occurs in tobacco-smokers, and is attended with defective central vision with usually no definite change in the fundus oculi.

Symptoms.—Defective sight is the chief complaint. There is progressive failure in both eyes, which, in the course of a few weeks, or months, may have become so marked that the patient can only distinguish $\frac{6}{36}$ or $\frac{6}{60}$, or No. 16 or 20 of the Jaeger reading-types. The sufferer is apt to mistake gold and silver coins; every object he looks at appears misty or as if in a fog, and he is apt to attribute it at first to a bilious attack; it is only after some weeks, finding his sight failing instead of

improving, that he comes for advice. He will often say that he can see people better and clearer as they pass him, than if he looks straight at them.

The *visual field* is almost characteristic. In every case of early tobacco amblyopia there is to be found a *central relative scotoma*; beyond this, any other change in the field is exceptional. The central defect is chiefly one of colour; green and red are the colours which cannot usually be recognised. Green is always lost before red. The scotoma varies in size and kind with the strength and amount of the poison taken, and so mainly depends upon the length of time that

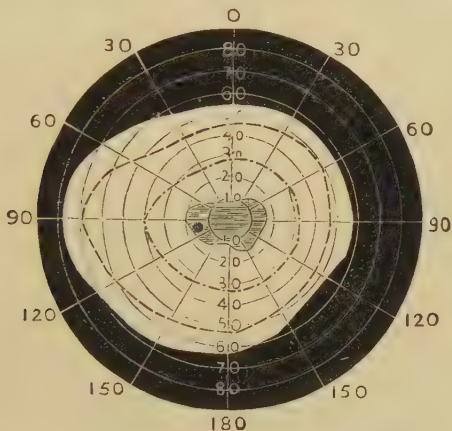


FIG. 66.—Visual Field in Tobacco Amblyopia (left eye).

has elapsed since the onset of the visual failure. In slight cases, where the disease has not been in progress for more than one or two months, a central scotoma for green only may be found, and it is often necessary to use a very small colour-disc (2.5 mm. in diameter) before it can be made manifest. In exceptional instances the green colour may be perceived in the central region, though less distinctly than in the visual field immediately around it. In a more advanced condition a definite central scotoma for green and red is readily found, and can be mapped out clearly on a chart as in fig. 66.

In severe cases, in which the vision has been impaired for twelve months or longer, the scotoma will be larger, and may

involve blue ; yellow is very rarely lost, though it is a frequent occurrence to find both yellow and white less distinct in the central region than just outside it. The field for green may be completely lost, or a small area may be left like an oasis in a desert to the inner or outer side. Peripheral contraction for colours, and even white, may be seen, and is suggestive of commencing optic atrophy (see fig. 66) ; it is more often witnessed in subjects who deeply indulge in the commonly associated poison, alcohol. The scotoma is never absolute, complete central blindness is never seen in this affection. Green and blue appear white, red looks a 'drab' or dirty brown colour, white is less bright by direct vision. The shape of the scotoma is generally found to be oval with its long axis transverse ; it encroaches more on the temporal than the nasal half of the field. Cases of unilateral colour scotoma are extremely rare.

The subjects of this affection are most troubled by bright light and by distant objects ; they can generally see better in twilight than in open day (day-blindness), and they find some help for this defect in the use of neutral-tinted glasses, by which the brighter rays are cut off. The peripheral portions of the field being good, they experience no difficulty in seeing surrounding objects ; they therefore differ somewhat in manner from patients who are suffering from diseases in which contraction of the visual field forms a prominent feature, such as advanced retinitis pigmentosa, optic atrophy, and chronic glaucoma.

The *ophthalmoscope* reveals nothing of importance in the condition of the fundus. Occasionally we find hyperæmia of the optic disc, and some enlargement of the retinal veins. In advanced cases there is sometimes a pale (atrophic) condition of the optic disc, beginning in the lower and temporal quadrant. The macular region often shows fine dull yellowish dots, which seem to occur too frequently to be mere coincidences.

The onset of the disease is very insidious ; in some cases hardly any other symptoms beyond the visual derangements are to be found, in others there may be frontal headache, nervousness, insomnia, and loss of appetite. A few cases of acute tobacco amblyopia have been recorded.

Etiology and pathology.—This form of amblyopia is now generally admitted to be produced chiefly, if not entirely, by tobacco intoxication. The subjects of it are generally males, at or beyond middle life, who have long been in the habit of smoking large quantities of strong tobacco. Unfortunately, many excessive smokers are also accustomed to free indulgence in alcoholic liquors, so that it is difficult to make out how far the defective vision may be due to the direct influence of alcohol. Mackenzie and Sichel long ago pointed out the deleterious effect upon vision of the excessive use of tobacco; the latter believed that any person smoking more than three-quarters of an ounce of tobacco daily would experience considerable defect both of sight and of memory. He mentions a case¹ of a man who, not content with smoking throughout the entire day, assumed the pipe at intervals during the night to soothe his wakeful hours. He became completely blind, but recovered his sight after total abstinence from smoking, combined with antiphlogistic treatment. Berry says that the consumption of one ounce of tobacco a week may cause amblyopia. On the other hand, people have smoked seven or eight ounces of strong tobacco every week for a long period of time without any symptoms of tobacco poisoning. In speaking of this affection Nettleship² says: ‘My own opinion, based on the examination of a large number of cases, is that tobacco is the essential agent, and that the disuse or diminished use of tobacco is the one essential measure of treatment.’ Many authorities, however, consider alcohol amblyopia to be a distinct affection. Uhthoff’s cases even point to alcohol being a more common cause of amblyopia than tobacco. In moderate quantities alcohol is considered by Jonathan Hutchinson and Berry to be an antidote for tobacco poisoning, but in excess to predispose to toxic amblyopia.

The quality of the tobacco and the form in which it is consumed are of almost as great importance as the quantity. Moist varieties of tobacco are more poisonous than dry, and the cheaper forms, since they contain more nicotine, are very toxic. Van Millingen believes that a solution of the nicotine

¹ *Annales d’Oculistique*, vol. liii. p. 122.

² *Diseases of the Eye* (1882), p. 217.

to cause tobacco amblyopia must be absorbed, and thus explains the fact that the Turks are immune, though smoking an enormous quantity of tobacco. This race takes precautions against any of the tobacco coming in direct contact with the saliva. Perhaps the fact that the Turkish people drink little or no alcohol may have something to do with this immunity.

The individual is more susceptible if the tobacco be smoked when the stomach is empty, or if the practice of inhaling be indulged in; but chewing is probably the most dangerous of all methods of consumption. Snuff, being a very mild form of tobacco, rarely, if ever, produces poisonous symptoms.

A few cases have been recorded of non-smokers who worked in tobacco manufactories, and who acquired all the symptoms of tobacco amblyopia.

Nicotine is not the only poisonous constituent of tobacco, many volatile oils, such as pyridine and collodine, being also present.

People suffering from insomnia, or from some mental distress caused by family or business troubles and the like, are more prone to fall victims to this malady than those in sound health and free from anxieties. It must not be forgotten, however, that at these times the quantity of tobacco consumed is often increased.

The pathological anatomy of toxic amblyopia has been, and still is to some extent, disputed. Various authorities have localised the lesion in the retina, the optic nerve, or the brain. It is now held by some that the intra-orbital portion of the optic nerve is in a condition either of true inflammation or of primary degeneration. Microscopical anatomy has shown a hypernucleation of the portion of the nerve known as the papillo-macular bundle, together with a connective-tissue hyperplasia. This may be a true interstitial retro-bulbar neuritis, or it may be of a sclerosing nature, similar to the changes found in a cirrhotic liver. The temporary nature of the affection, however, points strongly against this latter view; and it is only in those cases where the optic disc is pale, and where the vision is permanently injured, that sclerosis would seem to be present.

Others, however, consider that the primary lesion is in the ganglion cells of the retina. The activity of ganglionic nerve-cells is known to be very profoundly influenced by nicotine, and very definite changes have been found by Nuel and others in the ganglionic cells of retinae of patients suffering from toxic amblyopia.

The *treatment* consists in the removal of the cause and the improvement of the general condition of the patient. *Total and unconditional abstinence from all forms of tobacco and alcoholic liquors* should be insisted on. The patient will be greatly chagrined at the sudden cessation of these, to him, poisonous habits; he will beg hard to be allowed just one cigar and one glass of wine per diem; but he must not be humoured. Total abstinence is by far the most certain and speedy mode of cure; it should, of course, be combined with a tonic regimen. Nutritious food, plenty of exercise in the open air, sedatives at night if necessary to produce sleep, potassium iodide at first, followed by strychnine and iron internally, and similar remedies, are essentially helpful, and will generally restore the visual acuity, disperse the central scotoma for colours, and greatly improve the patient's general physique in the course of six to twelve weeks. Other drugs that have been used are pilocarpine in the form of hypodermic injections, nitroglycerin, and digitalis. Subconjunctival injections of cyanide of mercury, and of normal saline solution, have been found of little use.

Prognosis.—As a rule, the results of treatment are more pronounced in proportion to the rapidity of failure of sight, and to the shortness of the duration of the disease. In the majority of instances, if the treatment is properly carried out, a very favourable result may be anticipated, for vision is often completely restored. If there is no peripheral contraction of the colour-fields, and if the condition has not lasted longer than three months, complete restoration of vision is almost certain. In old-standing chronic cases, and especially where there is some pallor of the optic disc, the improvement is less marked, and perfect vision may not be re-established. Relapses are rarely met with, even if the habit be returned to in moderation.

Other Forms of Toxic Amblyopia.—Other substances, besides tobacco and alcohol, may produce toxic amblyopia.

Iodoform, whether taken by the mouth, or absorbed through the tissues when used as a dressing for a wound, may cause amblyopia. The symptoms closely resemble those of tobacco amblyopia, except that the central scotoma is often absolute.

Carbon-disulphide is largely used in indiarubber factories, and workpeople have been known to acquire toxic symptoms, including amblyopia, presumably by inhaling the fumes.

Nitro-benzol, quinine, ptomaines, tea, and many other substances may occasionally produce amblyopia.

Amaurosis is occasionally seen in young infants. The aimless movements of the eyes generally first attract the mother's attention, and it is then observed that the child takes no notice of a light. In such cases the fundus is sometimes normal, not unfrequently the discs have a greyish appearance, and their edges are a little blurred; later they usually become atrophic. In these cases there is sometimes a history of convulsions, and often there is evidence of inherited syphilis. The prognosis is absolutely unfavourable.

Amblyopia, from suppression of the image in one eye, is often found in cases of strabismus. (See Strabismus, p. 578.)

Hemianopia or **Hemianopsia**¹ is characterised by the loss of one half of the visual field. It usually occurs in both eyes, and is then indicative of some lesion at or beyond the optic commissure. When only one eye is affected, the line of separation between the part of the visual field which is lost and that which is retained is generally irregular; the affection is then the result of some lesion of the optic nerve in front of the commissure, or of the retina itself.

The majority of cases of hemianopsia affecting both eyes are either *right* or *left* lateral—that is, there is homonymous loss of the right or of the left half of the visual field in each eye—*homonymous hemianopsia*. The right half of each visual field of course corresponds to the left half of each retina, and

¹ The term *hemioopia* is used to denote the blind half of the retina, whereas *hemianopia* or *hemianopsia* expresses the defective visual field; acknowledging such, a lesion causing *right* hemioopia is one which produces *left* hemianopsia. It would cause less confusion to discard the word 'hemioopia' altogether.

vice versa. As a rule, the point of fixation lies in the part which retains its functions in both eyes, but occasionally the line of demarcation seems accurately to bisect it.

Varieties of Hemianopsia.

- | | |
|----------------------------|--|
| 1. Homonymous hemianopsia | $\left\{ \begin{array}{l} \text{Complete.} \\ \text{Partial.} \\ \text{Relative.} \end{array} \right.$ |
| 2. Temporal hemianopsia | |
| 3. Nasal hemianopsia | |
| 4. Altitudinal hemianopsia | $\left\{ \begin{array}{l} \text{Lesion almost invariably} \\ \text{at the chiasma.} \end{array} \right.$ |

In *temporal* hemianopsia the lesion involves the chiasma at the point of crossing of the inner half of each tract. Both nasal retinae become functionless, and each of the temporal visual fields lost. The disease is usually progressive, total blindness being reached as the result of implication of the uncrossed half of each tract. It is commonly associated with enlargement of the pituitary body, especially in acromegaly.

In *nasal* hemianopsia the lesion involves the fibres at the commissure which do not cross, so the temporal retina in one eye only is affected, and the nasal half of its field lost. It is difficult to conceive a lesion which could cause bilateral nasal hemianopsia, although undoubted cases have been recorded.

Altitudinal hemianopsia is loss of one or both upper or lower visual fields, and is likewise due to a lesion of the lower or upper portion of the chiasma.

These three varieties are rarely seen, and require no further comment.

The symptoms of homonymous hemianopsia.—The patient usually complains of sudden diminution or disturbance of vision. He sees only half of an object placed immediately in front of him.

In right homonymous hemianopsia there is marked inconvenience in reading. This is because, in order to read with fluency, it is necessary that words should be seen which are a little in advance of those which are being pronounced; when the right half of each visual field is lost, the words cannot be seen until their image falls on the yellow spot of the corresponding half of each visual field. The line of demarcation

between the sensitive and the inactive portions of the retina is usually vertical, either at, or just internal to, the yellow spot, leaving the macula in the seeing half. The transition from the one part to the other may be quite abrupt, or it may be gradual.

Fig. 67 represents a chart of the visual field for white, blue, red, and green, which was taken from the left eye of a patient suffering from left homonymous hemianopsia. By comparing this with the normal visual field represented in fig. 62, it will be observed that the whole of the outer part of the field is lost.

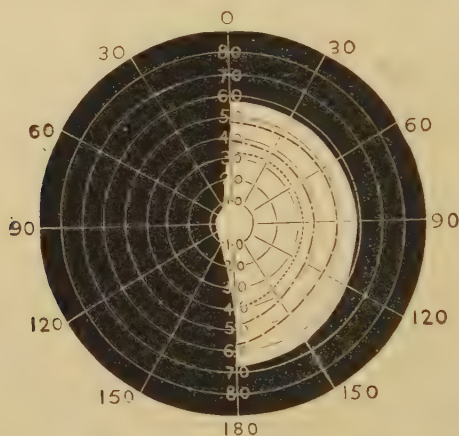


FIG. 67.—Visual Field in Temporal Hemianopsia (left eye).

The exact situation of the intracranial lesion-giving rise to hemianopsia is still somewhat uncertain. It may be situated anywhere in the *visual tract* (p. 238) beyond the chiasma. The lesion may be in the optic tract, the primary visual centres, the internal capsule, the optic radiation, or the cortex. If it be a cortical lesion or one occurring in the optic radiation, it may not be complete; that is to say, it may cause only *partial* homonymous hemianopsia; or, again, it may only involve the colour, or the colour and form centre (relative), without causing complete loss of sight: in the former case, colour will be lost, form retained; in the latter, a distinction between light and dark only will be made. It is exceptional to get hemiplegia with these cortical lesions. Alexia and optic

amnesia (see p. 334) are often present in cortical lesions. A lesion in the internal capsule, involving the posterior third of the posterior limb, theoretically would be attended with *complete* hemianopsia, but there is no clinical evidence that this is the case; hemiplegia would also probably be present. A lesion involving the primary visual centres has probably a similar result. A lesion involving the tract would give rise to the hemiopic pupil (see p. 600), a point of distinction from capsular lesions; the hemianopsia is usually complete; other symptoms of a basal lesion are also as a rule present.

Though the above course is mapped out for the visual path in which only a partial decussation is accepted as taking place between the optic nerve and its cortical centre, it does not explain the cause of crossed amblyopia; we have, however, sufficient pathological as well as experimental evidence to justify the assumption. Just as no centre in the cortex can be found controlling the action of the ocular muscles on the other side of the body, but only for their conjugate movements, so no cortical centre can be discovered having complete command over the vision of the opposite eye. Thus, there is an homonymous association in both the motor and sensory functions of the eye.

A lesion of the cortex attended with defective visual acuity as well as concentric contraction of the visual field of the opposite eye (*crossed amblyopia*) has been occasionally observed, and this, according to Gowers, is brought about by a lesion involving the angular gyrus.

Hun's case of partial homonymous hemianopsia, quoted by Swanzy,¹ is exceedingly valuable in that it gives an exact and limited area of the cortex for one quadrant of the visual field. The lesion, an old hæmorrhage, was found at the autopsy involving the lower half of the right cuneus; there was loss of the left lower quadrant of each field—*i.e.* the upper right quadrant of each retina was rendered functionless by that lesion.

In hysterical hemianæsthesia and in cerebral hemianæsthesia the unilateral defect is not confined to common sensibility; it involves also the special senses on the same side of the body

¹ *Trans. Ophth. Soc.* vol. xi. p. 183.

as the cutaneous anæsthesia; these are the nerves of taste, hearing, smell, and sight. Attention has been particularly called to the condition of vision by the observations of Landolt in certain cases of Charcot's at the Salpêtrière. He found—

(1) Normal ophthalmoscopic appearance of the fundus.

(2) Reduction of visual acuity to one half or more in the eye on the same side as the hemianæsthesia (crossed amblyopia).

(3) Concentric and general contraction of the visual field for white and for colours.

Mind-blindness, *i.e.* the perception of objects without recognition.—There are several forms of this condition, the symptoms varying with the extent and exact position of the lesion. As mentioned on p. 240, there is in all probability a higher visual centre, the memory centre, situated in the neighbourhood of the left angular and marginal gyri, and it is this part of the cortex, or its subcortical connection with the lower visual centres, which is at fault in this disease. The forms of mind-blindness may be divided into total mind-blindness and partial mind-blindness, the latter including word-blindness or alexia, difficulty in the power of reading or dyslexia, letter-blindness, figure-blindness, and note-blindness or amusia.

Total mind-blindness, or *optic amnesia*, is the result either of a cortical lesion involving the whole of the memory centre, or of a subcortical lesion completely cutting off this centre from the occipital perceptive centres. In the latter form, familiar visual objects, though not recognised, can be accurately described from memory. Hemianopsia is frequently present.

Partial mind-blindness, including *word-blindness* or *alexia*, *dyslexia*, *letter-blindness*, *figure-blindness*, and *amusia*, is in each case probably due to a partial cortical or subcortical lesion. In the subcortical forms there is no *agraphia*—*i.e.* the patient is able to write from dictation, though unable to read what he has written. In *dyslexia*, a few words only can be read at a time, the inability to continue being due neither to pain nor to visual dimness. A person may be able to read individual letters, but not words, resembling a child beginning to learn to read. Again, the power to read individual letters may be lost, though the

patient may still be able to read words. Word-blindness may be a marked symptom, though figures can be easily recognised. Finally, the visual recognition of musical notes may be lost (amusia), though words, letters, and figures are perceived and perfectly understood. Right homonymous hemianopsia is often associated with these forms of mind-blindness.

Congenital word-blindness—due, probably, to some mal-development of the higher cortical visual centres—is of importance from an educational point of view. Attempts should be made as early as possible to teach the child to read by means of individual letters, and in this way the expenditure of much time and patience will often be rewarded by considerable success. Some cases, however, resist all educative treatment, and the sooner they are recognised the better, in order that the children may be educated on other lines.

Night-blindness¹ has already been referred to as a symptom of pigmentary degeneration of the retina, and other lesions of the fundus. It is always an accompanying symptom of the epithelial form of xerosis conjunctivæ (see p. 101). Under certain circumstances, however, this affection is found to exist as a functional disorder. The characteristic symptom of functional night-blindness is that visual acuity, which is perfectly good in a bright solar or artificial light, becomes suddenly reduced when the sun gets below the horizon, or when the artificial light is reduced. The patient can see perfectly well during the day, but immediately after sunset, or when placed in a moderately dark room, the sight is so impaired that he has to grope about, and in some cases cannot find his way without the help of a guide; the visual field is not contracted; the fundus is normal in appearance; the pupil is sometimes half dilated; and there is generally some reduction in the range of accommodation.

The most common cause of night-blindness is the prolonged exposure of the retina to the action of strong brilliant

¹ Formerly the term *hemeralopia* was used to indicate this symptom, and *nyctalopia* the opposite condition of 'night-sight,' or day-blindness. An article, however, by Greenhill (*Ophth. Hosp. Reports*, X. ii. p. 284) shows that the true meaning of the words, according to their derivation and classical use, is the exact reverse of this. Under these circumstances it would create confusion to retain either term.

light. It is common among sailors who have made long voyages under a tropical sun, and soldiers after prolonged marches; painters and masons who have been employed on white buildings are also sometimes affected. This trouble is more prone to occur in persons whose vitality is lowered from insufficient or improper food, excessive work, and other causes. It is often associated with scurvy. A paper by Forster¹ would seem to prove that the affection often makes its appearance in hot climates without exposure to bright light, especially in districts where ague is common.

A form of night-blindness appears to be epidemic in Northern Russia. It has been thought to be miasmatic in origin.

Prognosis and treatment.—Night-blindness always improves under favourable conditions, although it sometimes evinces a tendency to recurrence. The first indication is to protect the eyes from all bright light. This may be done by keeping the patient in a feebly illuminated room, or by the use of very dark smoked glasses. The use of eserine drops (F. 16) is also advisable. A nourishing diet, and the use of iron, quinine, and other tonics, are valuable adjuncts. Ingestion of liver and cod-liver oil are also strongly recommended in this affection.

Snow-blindness, which is sometimes experienced by persons who have travelled over extensive tracts of snow, presents the same functional derangements as the night-blindness just mentioned, but there is usually congestion of the conjunctiva, with pain and photophobia. The pain in the eyes is often intense, the tears will stream out, and it is almost impossible to keep the eyes open. Instant relief is felt upon coming across a tract of bare ground devoid of snow. The symptoms may last a few days after the removal of the cause. It is prevented by the use of deeply tinted glasses, so arranged that the intense glare of the snow cannot reach the eyes.

Micropsia signifies a condition of sight in which objects look too small. Its occurrence is indicative of the rods and cones being pressed asunder, so that images formed on the retina coincide with fewer retinal elements. It is sometimes

¹ *Rec. d'Ophth.*, Oct. 1882, p. 577.

found in syphilitic retinitis. *Megalopsia*, the apparent enlargement of objects, and *micropsia* are sometimes found in hysterical amblyopia. *Metamorphopsia* means the apparent distortion of objects. To test whether this is present, a piece of paper with a number of parallel straight lines ruled upon it at distances of 2 mm. is placed before the patient, who fixes a black dot placed on the middle of the central one, and notices whether the lines appear parallel or bulge towards or away from the fixed point. The lines should be held both vertically and horizontally.

‘Eclipse’ Amblyopia.—Following each solar eclipse, a few cases of so-called ‘eclipse’ amblyopia have been recorded. A too daring observation with no protection, or a too prolonged gaze at the astronomical phenomenon even through a smoked or coloured glass, produces a very definite group of symptoms. A positive absolute scotoma is noticed immediately or very soon after the exposure; this often takes the form of a flickering or revolving scotoma. *Metamorphopsia* is usually present, *megalopsia* being more frequent than *micropsia*. Ophthalmoscopic examination often shows a normal fundus. There may, however, be definite changes, and of these the commonest is the presence of one or two bright shining spots in the macular region, with slight pigmentary disturbance. The amblyopia, though improving with complete rest, usually to some extent persists, and especially is this the case should a revolving scotoma be present. The *metamorphopsia* rarely completely disappears. Treatment consists of rest to the eyes, and the wearing of neutral-tinted glasses.

Malingering.—Simulated amblyopia, or simulated complete amaurosis of one or both eyes, is occasionally met with, but is less common in Great Britain than in countries where conscription is in force. It is found chiefly amongst those who wish to escape service; after injury, also, it is sometimes feigned with the hope of receiving compensation for damages. Amongst children, the desire to avoid school and lessons is sometimes the chief motive. It also occurs among nervous and hysterical young women.

One eye, usually the right, is generally complained of as being defective, the other eye being declared normal. Under

such circumstances the distant vision of each eye should be carefully tested, and the first statement as to the vision of the supposed amblyopic eye carefully noted. The deception may then be discovered in various ways.

1. *Von Graefe's method*.—Place a prism of 10° , base inwards, before the sound eye. If the patient be really using both eyes this will produce diplopia, and he will be observed to squint in order to correct this.

2. By means of Snellen's coloured test-types suspended in front of a window. The alternate letters are red and bluish-green—the exact complement of the red. The patient is told to read these with the good eye. Thus, suppose he reads the word FRIEND, of which FIN is green and RED red; then, by placing a bluish-green plane glass in front of the good eye he will see only the letters FIN if the other eye be amblyopic—for the red letters cut off all rays of light except the red, while these are cut off by the green glass which transmits none but green rays, therefore no light can pass through both glasses; if, however, he says he can still see the whole word FRIEND, he must be malingering.

3. Two very weak lenses may be alternately and repeatedly placed in front of the affected eye; if the patient believes that a succession of lenses is being tried, he will sometimes admit a gradual improvement, often up to normal vision.

4. By paralysing the accommodation of the good eye, or by placing a strong concave lens (-20 D) in front of this, and then directing the patient to read, we know that he can only do so with the affected eye.

5. Place a prism of 6° base upwards in front of the good eye so that the base corresponds to the middle of the pupil; this allows rays to pass into the eye above as well as through the prism, and *monocular diplopia* is produced. Now uncover the doubtful blind eye and simultaneously move the prism slightly upwards, so that rays can now enter the eye only through the prism; if diplopia still exists, it is binocular, and proves that vision is present in both eyes. If the supposed blind eye be once more covered, only one object will be seen.

6. The stereoscope, the perimeter, and other methods are also useful in the discovery of this kind of deception.

When amblyopia in both eyes is complained of, the mode of detection is more complicated, and requires greater tact on the part of the surgeon.

The refraction of each eye and the ophthalmoscopic appearance of the fundus being ascertained, the visual acuity of each eye should then be carefully recorded ; then by placing feeble convex or concave glasses in front of either eye, the patient will often betray himself by inconsistent replies. The visual field for white and for colours (p. 312) should then be tested. The nature of the answers to questions will here be also useful.

When complete amaurosis of one eye is asserted, it must be remembered that in such an eye the pupil would be dilated if the amaurosis had existed for a long time, and would not contract by the projection of a cone of light upon the cornea, supposing the opposite eye to be completely shaded from the light. In order to distinguish between dilatation of the pupil from blindness and that from atropine, we must bear in mind that the dilatation from atropine is usually greater than from amaurosis ; again, while in amaurosis a cone of light thrown upon the retina of the good eye would produce contraction of the sphincter pupillæ of the other, this would not be the case with dilatation from atropine.

CHAPTER X.

COLOUR-VISION AND ITS DEFECTS.

BY W. ADAMS FROST, F.R.C.S.

Normal Colour-vision.—Light is transmitted by means of transverse waves of ether ; the waves, however, in white or colourless light are not of one uniform type, but vary in length and rapidity of vibration. The waves of greatest length and slowest vibration are less easily refracted than the smaller waves with more rapid vibration ; hence, when a beam of solar light passes through a prism, it undergoes *dispersion*, or a separation into waves of different rates of vibration. Such of these waves as are capable of exciting the retina give rise to the sensation of *colour*, and the series of colours caused by the decomposition of light is called a spectrum. The largest waves which are capable of exciting the retina give rise to the sensation of red, the smallest to that of violet ; between these extremes are waves of gradually decreasing size and increasing rates of vibration from the red to the violet, and these give rise to various colours.

The colours of the spectrum, in their order, are—

(*Heat rays*), RED, Orange, Yellow, GREEN, Prussian Blue, Indigo, VIOLET (*chemical rays*).

These are the only ether waves capable of exciting the retina ; but at each end of the visible spectrum there are invisible waves. Those beyond the red are of still greater length, and are called heat rays ; and beyond the violet are smaller waves, which are called chemical rays. The difference, however, is of degree, and not of kind, for all the rays possess heat and chemical action, and the visible portion of the

spectrum owes its visibility, not to any difference in its physical character, but to the construction of our visual apparatus.

Light is reflected from objects in various ways. A surface reflecting light perfectly—a perfect mirror—would be colourless and invisible. A surface which reflects all the waves in the same proportion as they exist in white light, but reflects the waves irregularly, appears *white*. A surface reflecting no light, but quenching or absorbing all, would be invisible; if it reflected only sufficient light to render it visible, it would appear *black*, so that a black surface is visible only in consequence of the imperfection of its blackness. A coloured surface quenches some waves of colourless light, and reflects others; it is from the latter that we judge of the *colour* of the surface. Objects differ also in the manner in which they transmit light. Some are said to be opaque since no light rays pass through them, others are so perfectly transparent that they appear to transmit all light rays. Both terms are, of course, relative, since a layer of infinitesimal thickness of an opaque substance will transmit rays, and a great thickness of a transparent substance will obstruct them. Other substances transmit rays of a certain wave-length only; we have familiar examples in the red and blue glasses in railway signals.

The following are some of the principal terms which are applied to colours. Differences in *hue* or *tone* are those which exist between the various colours of the spectrum, as red, yellow, blue, &c. *Luminosity* or *brightness* indicates the total amount of light reflected by a coloured surface. Yellow is the most luminous part of the spectrum. A *dark* or *dull* colour, on the contrary, is one from which the total amount of light is small. All colours contain a certain amount of white light; even a spectrum, if thrown upon a white surface, receives some admixture of white light from it. Several terms are used to indicate the extent of this dilution. Thus the terms *saturated*, *deep*, *pure*, indicate that there is but little admixture of white; while the terms *pale*, *diluted*, *light*, indicate on the contrary a low degree of saturation, or much dilution with white.

Additional proof that white light is composite is afforded by

the fact that the colours of the spectrum can be re-combined, so as to form white, by means of a prism placed in the reverse position to that of the dispersion prism ; and that if the colours be painted on a disc (Maxwell's disc) in the same proportions as they exist in the spectrum, and the disc be rotated rapidly, a grey is produced, which approaches white in proportion to the purity of the pigments used and the accuracy with which the relative quantity of each colour has been measured.

In order to produce white, however, it is not necessary to employ all the colours of the spectrum ; a mixture of red, green, and violet will suffice for this purpose ; and because these colours cannot be produced by a mixture of others, they are called *fundamental* or *primary*. Any other colour of the spectrum (or a colour indistinguishable from it by the eye) can be produced by a combination of the primary colours on either side of it. In speaking thus of the mixture of colours it must be understood that coloured *light* is meant ; the effect of mixing *pigments* is different, because the pigments are impure. Thus a mixture of blue and yellow light produces white, but if blue and yellow pigments be mixed, green is produced ; this is because each of the pigments reflects some green light in addition to its own colour, so that by their union a green, more or less mixed with white, is produced.

Since white can be made by mixing the three primary colours, it follows that to each one of these there corresponds a *complementary colour* (*i.e.* a colour whose addition is required to make white), which is formed by a combination of the other two.

Thus the complement of *red* is green + violet = *bluish-green*.

 " " *green* is red + violet = *purple*.

 " " *violet* is red + green = *yellowish-green*.

In the same way, to each colour in the spectrum there is another which, added to it, produces white, and which is therefore said to be complementary to it. The relative positions of a colour and its complement are the same throughout the spectrum. Thus, if a colour be taken which lies to the right of red, *e.g.* orange, its complement will lie to the right of bluish-green, *viz.* blue, and so throughout the spectrum.

But here we must guard against an error. The centre of

each band of colour in the spectrum has a definite wave-length and rapidity of vibration peculiar to itself; and on either side the waves gradually approximate in character to that of the adjacent colour. By the mixture of two of the so-called fundamental colours a visual sensation is produced which is indistinguishable from that produced by the spectral colour that lies between them; this resemblance is probably due only to the imperfection of our colour-sense. Viewed in this light, the separation of the colours of the spectrum into fundamental and non-fundamental has a great value in relation to our perception of colours, but none in relation to their physical properties; in other words, its value is physiological rather than physical.

Purple seems to occupy an anomalous position in the scale of colours, for it is formed by the union of red and violet; yet these do not lie on either side of it, but at opposite ends of the spectrum. We have seen that beyond either end of the visible spectrum there are waves the rates of whose vibrations form a continuous series with those of the visible spectrum; so that from the large, slowly vibrating, ultra-red waves the rate of vibration gradually increases through all the spectral colours to the invisible ultra-violet rays; how far they extend in either direction we have no means of knowing, but there is no ground for supposing that they stop short at the point where we cease to be able to follow them. If we assume that beyond the violet these waves extend *ad infinitum*, their rate of vibration increasing at the same rate as in the visible spectrum, it is evident that at some distance beyond the violet they would form a series of waves which would stand in the same relation to the colours of the visible spectrum as a series of musical notes to their octaves. On this hypothesis, purple would take its proper place between the violet of the visible spectrum and the octave of the visible red, and the light of the sun would consist of an infinite series of waves, of which only a single octave would be capable of exciting any visual sensation.

The relation of the colour-sensations to each other and to white may be conveniently represented by a circle formed by all the colours of the spectrum, the red and violet being united by various shades of purple, and white being placed in the middle. Each colour and its complement would then lie at opposite

extremities of the same diameter, while white, which is formed by their union, would lie between them. Such a diagram, however, would give no indication of the proportion of each colour necessary; this can be done by altering the circle to a triangle, and placing the fundamental colours at the angles; a colour formed by the combination of any two will then be found on the line between them (fig. 68). We shall presently see that the purest primary colours do not give rise to the purest possible sensation of those colours, so that the sensations which we call red, green, and violet are indicated by a posi-

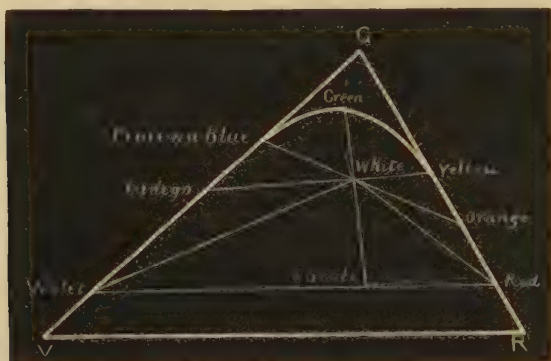


FIG. 68.—Relation of Complementary Colours.
(From Hermann; modified.)

tion a little removed from the angles. The position of the various colours, and of white, is so chosen that the latter always lies on the line connecting the complementary colours, and proportionately nearer to that one of which it contains most; and in the same way, any compound colour lies on the line between its components, and proportionately nearer to the one of which it contains most.

There is reason to believe that our sense of colour is very defective. In the first place, we know that there are waves on each side of the visible spectrum, which, although they possess no other difference, as far as we can ascertain, from those of the visible spectrum, yet excite no visual sensation. In the second place, the same visual impression is caused by colours that have no other claim to be considered as identical. Thus, a mixture of red and bluish-green, and one of yellowish-green

and violet, alike produce the sensation of white; yet a surface illuminated by the first would in a photograph come out black, while the second under the same conditions would appear very bright; by means of a prism, too, the mixtures could be resolved into their component colours. Helmholtz has compared our colour and musical senses, and shown how much more highly developed the latter is; for a good musical ear can not only assign to every note heard singly its true value, but can resolve a chord into the notes of which it is compounded, and even in the combined effect of an orchestra can recognise each component sound.

We shall presently see that the visual sensations produced by the fundamental spectral colours, although the purest ever experienced, are under the ordinary conditions of vision less pure than certain *subjective* sensations of these same colours.

We must first glance at the physiological relation that exists between complementary colour-sensations. If any bright colour be looked at steadily for about half a minute, and the eye be then directed to some white or grey surface, an *after-image* is seen, whose colour is complementary to that of the surface originally looked at. If the complement to one of the spectral colours is looked at in this manner, the hue in the after-image is brighter than the corresponding hue in the spectrum, and gives rise to a purer sensation of that colour than can be obtained in any other way.

To construct a theory of the mode in which colours are perceived, which should explain the relation of the fundamental to the other colours, the physiological relation of the complementary colours, and the mistakes made by those who are colour-blind, was a problem which occupied physicists and physiologists during the first half of the nineteenth century. In 1800, however, the mighty intellect of Thomas Young had already formulated such a theory; but as it was in advance of the accepted physiology of his day, it lay dormant and forgotten until revived and slightly modified by Helmholtz, when it was found not only to explain nearly all the phenomena associated with our perception of colours, but to be in strict accordance with many facts which have been discovered and theories which have been accepted since it was first constructed.

Young's theory was, that in the eye there existed three sets of fibres, each of which was excited by one of the fundamental colours and by the non-fundamental colours near it in the spectrum, so that each fundamental colour excited only one set of fibres, but a non-fundamental colour excited the fibres corresponding to the fundamental colours on each side of it. Thus red and green would each excite one set of fibres only, while yellow, which lies between them in the spectrum, would excite both the red and green fibres; this explains why the same visual sensation is produced by a pure spectral yellow, and a yellow produced by mixing red and green. This theory, however, although capable of accounting for most of the facts connected with colour-vision, leaves a few unexplained—for instance, the fact of the subjective sensation of the after-image of bluish-green (the complement of red) being more intense than that caused by the primary sensation of the purest red in nature, namely, that of the spectrum; it fails to explain, also, why those who are blind to red confuse certain shades of red and green, for according to it pure red would excite no visual sensation at all. To meet these difficulties, Helmholtz modified the theory somewhat.

Young-Helmholtz Theory.—This modified theory is as follows: That there exist, as assumed by Young, three sets of nerve-elements corresponding to the three fundamental colours, but that each of these colours, in addition to exciting its own special elements, excites also, but in a much less degree, the others. The effect of the various colours of the spectrum in different sets of elements can be conveniently shown by the accompanying diagram (fig. 69). The curves 1, 2, and 3 represent respectively the nerve-elements corresponding to red, green, and violet; the height of the curve at any point is in proportion to the degree in which it is stimulated by the colour indicated below by a letter. The sensation of white is produced by the maximum stimulation of all three.

According to this view, red not only stimulates strongly the red elements, but also to a slight extent those for green and violet. If, then, we could eliminate the action of the two latter, we should get a purer sensation of red; this can be done by gazing at a colour formed by the union of green and

violet, viz. bluish-green; the elements corresponding to green and violet then become fatigued, and the complementary red of the after-image consequently appears more vivid than the purest red with the eye in its natural condition. The Young-Helmholtz theory is now very generally accepted. There are, however, several rival theories, most of which are merely modifications of it, and need not detain us; but what may be termed the photo-chemical theory of Hering must be briefly noticed.

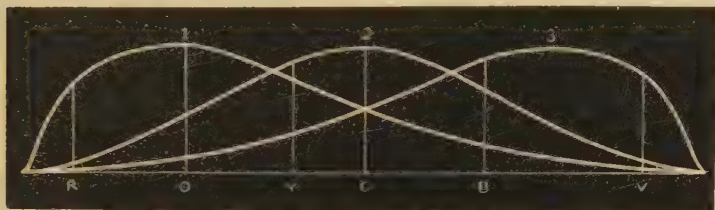


FIG. 69.—Holmgren's Diagram of Normal Colour-vision.

Hering's Theory.—It was discovered some years ago that there existed in the retina a substance, which received the name of *visual purple*, upon which light under certain conditions acted chemically, producing a kind of photograph of external objects. Hering assumed that there are three substances, each of which is acted on chemically by two colours but in opposite ways, the one colour causing disintegration of the substance, the other building up; and he accordingly designated each as a *dissimilation* or an *assimilation* colour; these substances he considers as corresponding to the following pairs: (1) Red and green, of which red is the dissimilation and green the assimilation colour. (2) Blue and yellow—of these he is uncertain which to consider as dissimilation and which assimilation. (3) White and black, of which white is the dissimilation colour.

According to this view, white and black are considered as specific colour-sensations, and not as expressing the combined effect of all colours or the absence of light.

The foundation for Hering's theory is the assumption that the visual purple plays an essential part in vision; but this is by no means proved. That prolonged exposure to light of a delicate membrane like the retina should produce some

changes is not surprising ; but if such changes were essential to vision they would surely be most marked where vision was most acute—viz. at the yellow spot—but here the visual purple is absent. This theory, however, does explain a fact that the Young-Helmholtz theory does not account for—viz. that a complementary after-image is seen when the eyes are closed.

Edridge-Green's theory of colour-vision differs radically from those just described. Both these rest on the assumption that normal colour-vision is trichromic ; that is, that there are only three primary colour sensations—red, green, and violet—and that all others are produced by combinations of these. This assumption rests on the physical basis that these primary colours mixed in certain proportions produce white, and that all colours can be produced by an admixture of the primary colours.

Edridge-Green, on the other hand, maintains that all the colours of the spectrum are capable of producing primary colour sensations, and that, therefore, from a psychical point of view there is no distinction between primary and other colours ; and he makes the number of colour bands seen in the spectrum the chief basis for the classification of the degree of colour-vision.

Assuming that the facts are correct, we have here an excellent method for indicating the different grades of colour-vision ; but it does not appear to us in any way to explain colour-vision and its defects, while the perception of the various kinds of physical series given by Edridge-Green seems to present little or no analogy with that of the spectrum.

The retinal elements which are essential to colour-vision are probably the cones, for they are most abundant at the yellow spot where colour-vision is most acute, and more sparsely scattered at the periphery where colour-vision is very defective ; and in animals whose habits are nocturnal the cones are absent (Schultze).

The periphery of the visual field is blind to all colours, the field for green being the smallest. It has, however, been said (Landolt) that, if colours of great intensity be employed, they can be recognised quite up to the periphery of the field. Since the colour of objects depends on the light reflected from them,

it will necessarily vary with the incident light. If, for example, this contains no red rays, objects which in ordinary light are red will appear colourless. If the amount of light reflected is sufficiently bright to stimulate all the three sets of elements to their maximum extent, the sensation of white is produced whatever may be the colour of the reflecting surface by a feebler illumination. A colour which has the greatest intrinsic brightness—*i.e.* reflects the most light—most easily passes into white ; in this respect yellow takes the lead. For the same reason colours of a low degree of saturation—*i.e.* containing much white light—differ less from each other in appearance than more saturated colours ; this fact has an important practical bearing in testing the colour-vision.

Congenital Defects of Colour-vision.—It has long been known that persons are occasionally met with who, although possessing normal sight in all other respects, fail to see any difference between colours which to other people are totally distinct ; they are therefore said to be *colour-blind*. The earliest published case of colour-blindness is that of a shoemaker in Cumberland named Harris.¹ In 1794 Dalton discovered his own defect, and mentioned other cases. But it is only within the last thirty years that attempts have been made to ascertain the frequency of colour-blindness ; and the result of the examination of large numbers of persons, in this country, on the Continent, and in America, has been to establish the fact that, although it is a rare defect among females, the proportion of colour-blind persons among males whose sight is otherwise normal is not less than 4 per cent.

That the percentage should be so high seems at first sight almost incredible, but the defect is not only one that can easily be concealed by the subject of it, but one of which he may be himself entirely unconscious. In early life we learn to associate the names of certain colours with the names of common objects ; thus we learn very early that grass is green, the clear sky blue, and that a soldier wears a red coat ; a child who has normal colour-sense soon learns to recognise similar qualities in other objects and to call them by the same name, whilst one whose colour-vision is defective learns by

¹ *Phil. Trans. of Royal Soc.* 1777.

heart the colours of common objects, without recognising the true distinctions. As he grows older he is puzzled to find other objects designated by the same epithet; if he attempts to name the colour of unfamiliar objects he makes mistakes, for which he is laughed at, and he probably thinks no more about the matter, but does not again commit himself to giving a name to a colour. Except in certain employments, it is very seldom that one is called upon to name a colour or to match two coloured objects; and it must, moreover, be borne in mind that the colour-blind do not confuse all colours, but only a few, and not all shades of those; so that a man may easily reach adult life without suspecting his defect himself, and still more easily without exciting any suspicion of it among his friends. The case of a woman is somewhat different. Except in the lowest grades of society, it would hardly be possible for a woman who was colour-blind long to conceal her defect; but among women the defect is, as we have said, extremely rare.

Throughout this chapter it must be understood that we are speaking of a congenital, not of an acquired, defect; in many morbid conditions the loss or impairment of colour-sense is an important symptom, and in some toxic forms of amaurosis—notably that due to tobacco—the loss of colour-vision over a limited area of the visual field is a characteristic symptom. These acquired defects will, however, be more appropriately considered under the diseases in the course of which they occur.

Degrees of colour-blindness have been variously classified according to the different theories of colour-vision. Up to the present time the theory of Young, modified by Helmholtz, has held the field, and this is generally used as the basis of classification. As the essence of this theory is the existence of separate nerve-elements for each of the three fundamental colours, so defective colour-vision is explained by the absence, or impaired function, of one or more of these sets of elements. Thus we may have—

A. **Total colour-blindness** (achromatopsia), in which there would be merely one set of fibres capable of excitation, and therefore all differences of colour would make themselves known according to the degree of excitation they caused, and would be perceived only as various degrees of brightness.

Total colour-blindness is, however, extremely rare, and need not be further considered here.

B. Complete blindness for one of the fundamental colours (partial achromatopsia). Thus we may have—

- i. Complete red-blindness ;
- ii. „ green-blindness ;
- iii. „ violet-blindness (or blue, according to Maxwell).

C. Incomplete blindness for one of the fundamental colours.

D. Incomplete blindness for all three.

The two latter may be conveniently classed together as feeble chromatic sense.

Among pronounced cases of colour-blindness—Group B—red-blindness is the most common, while violet-blindness is very rare.

The red- and the green-blind possess, as we shall presently see, many points of resemblance, and are equally important in cases where the competence of the subject to distinguish signals is in question. For these reasons, those coming under B i. and B ii. are sometimes classed together as ‘red-green-blind,’ as they would necessarily be if Hering’s theory were taken as the basis of classification.

We have seen that, according to the Young-Helmholtz theory, each fundamental colour, in addition to exciting the special fibres corresponding to it, excites also, but in a less degree, the other fibres; it is evident, therefore, that the absence of one set of fibres must alter the perception not only of the fundamental colour which most powerfully excites it, but also of those which excite it in a less degree. This will be made clearer by a reference to fig. 70, which is a reproduction of fig. 69 with the curve 1 omitted, and therefore represents the colour-vision of the red-blind. The sensation of white is now produced by the excitation of *two* instead of *three* sets of fibres. *Red* will excite the fibres for green, and very slightly indeed those for violet; therefore, the sensation of green will be produced. Since the amount of excitation of each set of fibres is comparatively slight, the colour will appear to be lacking in brightness; but as the stimulation is confined almost entirely to the one set of fibres,

there will be little appearance of admixture with white : therefore the red will appear as a saturated green of low intensity.

Red, orange, yellow, and green will obviously produce very similar sensations, but the green will be the most intense—*i.e.* the brightest, and at the same time the least saturated—

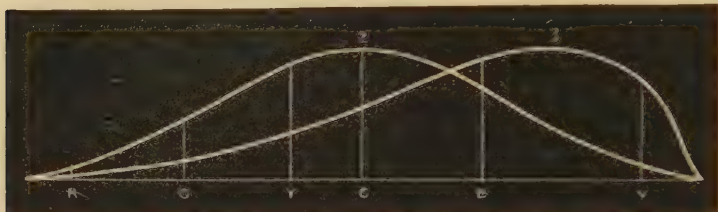


FIG. 70.—Colour-perception of Red-blind. (Holmgren.)

or containing the greatest amount of white. A red-blind person, therefore, would distinguish red and green only by their difference in brightness ; if the two appeared of the same intensity to the normal eye, the green would appear the brightest to the red-blind ; and if given several shades of red and green, and told to find two—one of each colour—which appeared to him alike, he would match a dark saturated red with a bright green. It is evident, however, that yellow and blue would give rise to totally different sensations, and would therefore not be confounded by him.



FIG. 71.—Colour-perception of Green-blind. (Holmgren.)

In the same way, for the green-blind curve 2 is omitted (fig. 71).

Red will be a saturated colour of low intensity ; yellow will be slightly more intense, and whiter.

Green is composed of nearly equal parts of the two fundamental sensations which in the green-blind produce white by

their combination, but being of low intensity is equivalent to grey. The impression produced by a yellowish-green, however (between yellow and green), would not be easily distinguished from a yellowish-red (scarlet) between red and orange, except that the latter would appear brighter. Hence the green-blind will not unfrequently match a scarlet with a yellowish-green which to the normal sight is much brighter.

Violet-blindness is extremely rare, and not of so much practical interest as the preceding varieties, as it produces no confusion between red and green, which are the colours used in signalling.

Edridge-Green, on the other hand, classifies the colour-vision of individuals according to the number of colours they see in the spectrum, and the length of the spectrum as seen by them.

Thus, those with very good colour-vision see seven bands in the spectrum; but a very large number do not make a distinction between Prussian blue and indigo, thus reducing the number to six. Failure to distinguish orange as a separate hue between red and yellow reduces the number to five; but this, as well as the reduction to four colours (by failure to distinguish between blue and violet), is, according to the author of the theory, of no practical importance, provided that the red end of the spectrum is not shortened, for these persons never make mistakes between a red, green, and a white light.

When only three colours are seen in the spectrum—red, green, and violet—the case is different. According to Edridge-Green, although the subjects of this defect do not confuse a red and green light under ordinary circumstances, yet under unfavourable conditions of light and atmosphere they pass into the condition of those who see only two colours in the spectrum—the red at one end and the green at the other, merging into one another directly or separated by a neutral band. These latter, it is said, see the difference between red and green as a matter of degree only, and easily make mistakes.

A very serious defect is shortening of the red end of the spectrum, because a red light is under these conditions rendered invisible by slight haze or fog, when it would be easily seen by one possessing normal colour-vision.

Methods of Testing Colour-vision.—The practical importance of being able with certainty to detect defective colour-vision lies in the fact that the lives of many may be sacrificed by one man mistaking a red for a green signal. Hence it would seem at first sight that the best test would be to show the examinee red and green signals in succession, and ask him to name the colour. Such a test would, however, be inefficient for several reasons. We have seen that the red- and green-blind do not confound all reds and greens, but only certain shades of these colours; given a red and green, which to the normal eye appear of equal brightness, the red will appear the brighter to the green-blind, and the green to the red-blind. Seeing the two lights in quick succession, a man who is red- or green-blind may recognise the difference between them, and name them correctly; possibly he is unaware of his defect, and believes that he recognises the true difference between them. But this is not sufficient; he may in clear weather, and at a known distance, recognise a signal correctly; but if that which is to him the brighter light is obscured by steam or mist, how is he then, with no standard of comparison, to recognise it? At sea, too, a fresh difficulty is interposed by the fact that the distance of the light is unknown. The use of coloured lanterns, or a lamp with coloured glasses and diaphragms, so that signal lamps at different distances can be represented, is interesting as a confirmation of other tests, but is too tedious and complicated for a first test.

Many lanterns have been devised which show coloured lights under the same optical conditions as those under which their recognition is necessary by engine-drivers and others. Some of these are provided with iris diaphragms, which enable the light to be increased in size at a rate corresponding to any given speed of a train. Nettleship, in conjunction with Brailey, made some experiments on colour-blind people, from which the following conclusions were arrived at: ¹

1. When red and green are shown together, they are often correctly distinguished if well within the maximum distance.

2. If white and red, or white and green, are shown together, they are always seen to be different, but are often wrongly named.

¹ Appendix C to Report on Colour-Blindness, *loc. cit.* p. 206.

3. By using various shades of smoked glass it is possible to make the white light indistinguishable from either red or green to the colour-blind.

4. When only one light is shown, whether white, red, or green, it is often, but by no means always, wrongly named.

A test should be sufficiently rapid to enable a large number of persons to be examined in a short space of time, and should not require the correct naming of colours. Although few with normal colour-sense could have any difficulty in naming such shades of red and green as are employed in signalling, it is otherwise with the more diluted colours which are used in most tests; hence ignorance of colour-names may easily cause the rejection of a candidate whose colour-perception is normal if the naming of the colours be insisted on. Another objection to the naming of colours is that, if the number of test-objects is small, the candidate may have been taught to name them correctly. It is, of course, essential that it should be a real test—*i.e.* that it should allow no one with defective colour-vision to pass, or condemn any with normal colour-sense.

Holmgren's wools constitute a test which fulfils these requirements better than any other. This method consists in making the observer pick out from a heap of wools those which seem to him to be the same colour as one given to him. Skeins of wool have been chosen as the test-objects, for the following reasons amongst others: the colours are purer, and the surface reflects less white light than pieces of paper, glass, or other stiff material. They can be obtained in any variety of colour, and are uniformly coloured throughout.

The tests are three in number: the first will detect all those who have any defect of colour-vision, the others will determine the nature of the defect.

The wools must be placed on a flat surface, on a white cloth, in good daylight.

Test I.—In the first test a skein is taken as the colour, which is a pure green rather freely mixed with white; it is represented very accurately in the colour-plate opposite page 356.¹

¹ Great pains have been taken by Messrs. Lebon & Co. to reproduce the colours of the wools in the plate, but it is difficult or impossible to do so

The heap of wools should consist of: (1) A variety of shades of green of the same character as the test-colour; other greens also may be added, such as blue-green, but they make the test longer, and do not add to its efficiency. (2) Various shades of the confusion-colours (1-5, in the plate), consisting of greys, drabs, yellows, rose, and salmon colours, all freely diluted with white.

The test-colour is shown to the examinee, and he is told to look at the heap and to pick out from it those skeins which appear to him to be the same colour as it, it being explained that they may be of lighter or darker shades.

With people of low intelligence, and with children, it is a good plan for the examiner to go through the test himself to show how simple a matter it is; if the number of wools is sufficient, and they are properly mixed afterwards, this gives no unfair assistance to those whose colour-vision is defective.

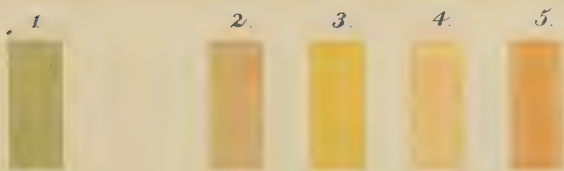
The directions may be given to a large number as many as can conveniently see—at the same time, and then each one is told to step forward in turn and go through the test. Those with normal colour-sense, as a rule, pick out the correct wools quickly and without hesitation; those who have any defect choose their wools in a slow, hesitating manner, and with them select one or more of the confusion-colours, and miss some of the greens. Any who choose a confusion-colour, or show a genuine doubt as to whether they should choose one—even though they reject it—should be subjected to the second test. Those who pick out all the correct wools and no confusion-colours may be considered to possess normal colour-sense. After a little experience one learns to recognise those who suspect their own deficiency, by their general behaviour; they, as a rule, hang back, and watch the performance of others with great care; when their turn comes they are most laboriously careful, taking up each skein and looking at it minutely. The inexperienced examiner may, however, if he trusts to general behaviour, occasionally mistake nervousness

accurately. The plate is intended to represent the wools only, and must on no account be itself used as a test, as some of the compound colours are composed differently from the dyes used for the wools.

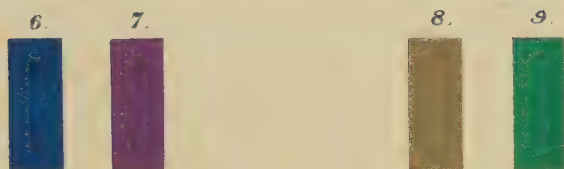
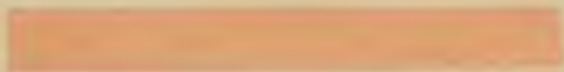
Plate XIV.

TESTS for COLOUR BLINDNESS.
(After Holmgren.)

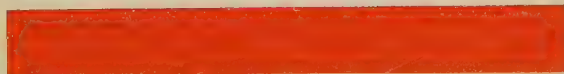
I.



IIa.



IIb.





or stupidity for defective colour-sense; the difficulties arising from the former can always be overcome by tact and patience.

The object of Test I. is to separate those whose colour-vision is normal from those in whom it is defective: the nature of the defect is determined by the following test.

Test II.—A rather pale but bright shade of purple (rose) is taken as the test (II. a represents it fairly well, but is a little too dull). The heap of wools consists of (1) various shades of purple, (2) various shades of the *confusion-colours* (6-9)—blues, violets, greys, and greens.

Purple, being composed of red and violet or blue, is to the red-blind identical with the two latter colours. For the green-blind a combination of red and violet produces white or grey, and green (*vide* G, fig. 71) produces a similar effect, but less intense.

Therefore—

The *red-blind* chooses *blue* and *violet* (6 and 7);

The *green-blind* chooses *grey* and *bright green* (8 and 9).

He who, having failed in Test I., chooses only purples, has a weak chromatic sense—*i.e.* he may have any of the defects enumerated under C and D on p. 351. There is no practical advantage in endeavouring to distinguish between these. The examination may close here; but the following may be used to confirm the result in those who have failed in the preceding tests.

Test III.—A bright red, such as is employed in signal flags (II. b), is used as the test-skein. The confusion-colours are dark and light shades of green and brown (10-13), which should be rather darker than 10, or olive colour.

The red-blind chooses a green and a dark brown (10 and 11), the latter being a combination of greenish-yellow with black.

The green-blind chooses a green, brighter to the normal eye than the red, or lighter brown (12 and 13).

A convenient arrangement of Holmgren's wools has been adopted by Thomson of Philadelphia, and is shown in the woodcut (fig. 72). The skeins, instead of being thrown promiscuously on the table, are hung by one extremity from a bar, and to each skein is attached a number, which is, however,

concealed from view while the instrument is in use. The skeins, of which there are forty, are numbered in the following manner: The test-colours are Nos. 1, 21, and 31. Nos. 1 to 20 consist alternately of colours matching the test-colour and the confusion-colours. In the same way with Nos. 21 to 30 in the second test, and with Nos. 31 to 40 in the third test. The wools should be arranged in an irregular order on the bar (not as they are shown in the figure), and the numbers of the skeins which the examinee chooses in each test are noted;

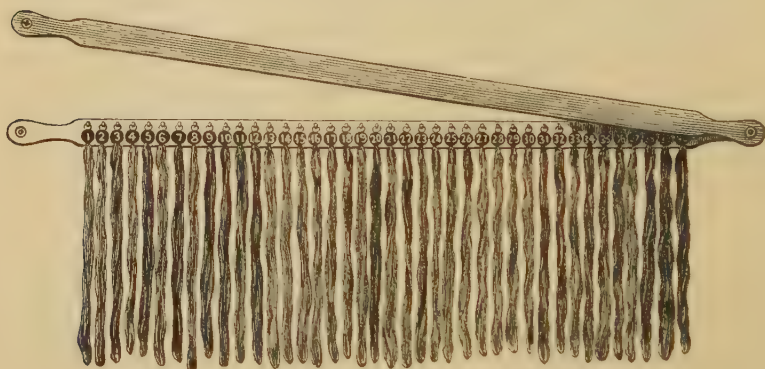


FIG. 72.—Thomson's Arrangement of Holmgren's Wools.

if his colour-vision is normal, these will of course consist only of odd numbers.

Edridge-Green does not admit the adequacy of Holmgren's test, but maintains that some individuals who pass this test without making a mistake can be demonstrated to be colour-blind by the lantern test.

The Frequency of Colour-blindness.—It has been ascertained by the examination of large numbers of people, chiefly by Continental and American observers, that the number of colour-blind persons is on an average a little over 4 per cent. of the male population. In consequence of this discovery, the governments of various countries have been urged to make compulsory the testing of the colour-vision of railway *employés* and seamen; and in this respect Holmgren in Sweden, Joy Jeffries in America, and T. H. Bickerton in this country, have been especially active.

It was felt that it was extremely important to ascertain whether the frequency of colour-blindness was as great in this country; and the Ophthalmological Society of Great Britain accordingly, in 1880, appointed a committee, of which the writer was a member, to investigate the subject. The following are some of the results.¹

The total number examined was 18,088; of these, 16,431 were males and 1,657 were females.

Of the males, 1,785 were taken from classes which it was suspected might contain an exceptionally high percentage of colour-blind—these were imbeciles, deaf-mutes, members of the Society of Friends, and Jews: all, except the first, gave a percentage above the average.

Deducting these, there remain 14,646 males, and of these 4·16 per cent. had defective colour-vision, in 3·5 per cent. the defect being of the pronounced character classified under B on p. 351. Comparing different classes of society together, colour-blindness would seem to diminish in proportion as education improves. Thus, among the schools of the poorer classes in Dublin² the average of pronounced cases was 4·2 per cent. Among the London metropolitan police and schools of the same rank, it was 3·7 per cent. In middle-class schools it was 3·5 per cent. Among medical students and the sons of medical men it was 2·5 per cent. Among the boys at Eton it was only 2·46 per cent.

Although, however, the frequency of the defect diminishes with the education of the class, the education of the individual has no tendency to remove the defect; this is shown by the fact that there was no appreciable difference between the children and adults in the same class, and is consistent with the history of individuals who have known themselves to be colour-blind. Thus, Dalton discovered his defect in early life, and always took great interest in comparing his ideas of colours with those of other people; yet he remained colour-blind to

¹ The Report of the Committee is published in the *Transactions of the Ophthalmological Society*, vol. i. p. 191.

² These are not included in the grand total, which is for England only. The results were obtained by Swanzy from an examination of 2,859 male children.

the same extent throughout his life ; and the same has been recorded of others. Indeed, there is no case on record in which a person proved to have had congenital colour-blindness has succeeded in removing the defect.

There can be little doubt that practice in distinguishing between colours, continued through several generations, would have a tendency to produce higher development of the perceptive elements, while want of practice continued in the same way would lead to their degeneration. In this way may probably be explained the great rarity of colour-blindness among women (only 0·4 per cent. of the number examined, and those for the most part slight cases), and its comparative frequency among the Society of Friends (5·9 per cent. of males and 5·5 per cent. of females).

The defect, having once appeared, would have a tendency to be handed down to posterity, especially if intermarriage took place within a class in which colour-blindness was especially frequent. In connection with this, it is interesting to note that the daughters of a colour-blind parent, although not exhibiting the defect themselves, may yet transmit it to their children. Thus, in an instance which came under the writer's own observation : a colour-blind parent had seven sons, all of whom were colour-blind except the youngest, and three daughters, none of whom were colour-blind ; but the son of the only daughter who married was colour-blind.

CHAPTER XI.

THE CRYSTALLINE LENS.

ANATOMY AND PHYSIOLOGY—CONGENITAL DEFECTS—LENTICONUS—COLOBOMA—CATARACT—VARIETIES OF CATARACT—ETIOLOGY AND PATHOLOGY—SYMPTOMS AND DIAGNOSIS—TREATMENT—NEEDLE OPERATION—LINEAR OPERATION—SUCTION—ARTIFICIAL MATURATION—FLAP OPERATION—VON GRAEFE'S LINEAR AND ALLIED OPERATIONS—ACCIDENTS AND IMMEDIATE COMPLICATIONS—AFTER-TREATMENT—REMOTE COMPLICATIONS—OPAQUE CAPSULE—DISLOCATION OF THE LENS.

ANATOMY AND PHYSIOLOGY.

The **crystalline lens** is a transparent biconvex solid body, enclosed in a transparent elastic structureless membrane—the lens capsule. In front of the lens is the iris. When the pupil is contracted the iris rests on the anterior surface of the lens, and is pushed somewhat forwards by it; when the pupil is fully dilated, no part of the lens is in contact with the iris; while in intermediate conditions a corresponding extent of the surface of the iris is in contact with the lens. Behind, the lens rests entirely against the vitreous humour, lying in a depression, the *fossa patellaris*.

When the accommodation is relaxed, the convexity of the lens is greatest posteriorly (fig. 73, A); during the act of accommodation, the convexity of the anterior surface is greatly increased, and that of the posterior very slightly, if at all, so that the curvature of the two surfaces is then very nearly equal. (See Accommodation.)

The measurements of the lens in adult life are from 8 to 9 mm. across, and 4 to 5 mm. from before backwards. The refractive power of the lens is not uniform throughout, it decreases slightly from within outwards; the mean refractive index for all areas is 1.4545.

By a series of admirably conducted experiments made upon lenses in each decade of adult life, Priestley Smith¹ has found that the average weight of the lens continually increases, the increase

¹ *Trans. Ophth. Soc.* vol. iii.

being at the rate of about 1·5 milligramme each year; also that the volume of the lens increases continually, at the rate of about 1·5 cubic mm. each year.

Histology.—The *capsule* is thickest in front, and diminishes towards the posterior pole. The part which covers the front of the lens (anterior capsule) is lined with a single layer of hexagonal, transparent, granular-looking, epithelial cells, each having an oval or a spherical nucleus. This layer of cells is of great physiological importance; from it the lens fibres are probably derived. It governs the nutrition of the lens by promoting proper osmosis between the lens tissue and the lymph in the anterior chamber (Leber). In this nutritive function it is probably greatly assisted by the ciliary processes, which are in close contact with the suspensory ligament just before it reaches the capsule. Entering at the equator, the nutritive fluid passes between the lens fibres and is thought by some to be collected at the anterior pole, whence it passes back to the equator to leave the lens close to its entrance.

The part which is behind the lens (posterior capsule) has no epithelial lining of this kind; it is in close contact with the lens-substance in front and with the vitreous humour posteriorly.

The *substance of the lens* is made up of lens fibres and interstitial substance. The fibres are bandlike structures, their cross-section showing a compressed hexagonal shape. The peripheral fibres are smooth, the intermediate and central ones have serrated edges; these do not interlock, but the apices of the serrations are in contact so as to leave interspaces for the interstitial substance. Oval nuclei are found in the peripheral fibres, but are absent from the central ones. The fibres extend between the anterior and posterior surfaces of the lens, and are arranged in concentric lamellæ parallel to the surface. Each lamella consists of a single layer of lens fibres joined at their broad surfaces. Their extremities are slightly enlarged. At the two surfaces of the lens these extremities are united together by three raylike structures, which in the early stage of cataract can often be seen by focal illumination, in the form of white lines diverging from the poles to the circumference at equal angles. In the natural state these sutures contain a semifluid homogeneous interstitial cement substance. A similar substance is contained between the lamellæ, and, in smaller quantity, between the fibres of each lamella (Klein). In this cement substance there exist certain channels, from which fine canals extend between the fibres of the lamellæ. These probably have an important bearing on the changes in the shape of the lens during accommodation, and in the nutrition of the organ (Otto Becker). The central portion of the lens is of

firmer consistence than that of the periphery; hence the central portion of the lens is called the *nucleus* and the peripheral portion the *cortex*. The comparative size of the nucleus to the cortex increases with age. In young subjects the lens substance is soft and easily broken down; at the age of twenty its nucleus has become harder and well formed; in later years it increases at the expense of the cortex, the elasticity of the lens consequently diminishing, its consistence becoming firm and its form less convex. Finally the cortex completely disappears.

The **suspensory ligament of the crystalline lens** (zonule of Zinn) is composed of a large number of elastic fibrillæ arranged in interlacing bundles and extending from the region of the ora serrata to the equator of the lens, passing over the ciliary processes. It has a multiple origin, some of its fibres being attached to the hyaloid membrane, others to the minute serrations of the ora serrata, others to the ciliary processes, while some appear to be continuous with the epithelial cells of the inner layer of the pars ciliaris retinæ. The bundles vary considerably in length, and may be divided into those composed of short and those of long fibres. The former serve the purpose of strengthening the latter, and are of two varieties: one set passes from the ciliary processes to join the long bundles; the other passes between the processes, joining them together. The ligament so formed passes as a whole forwards to the anterior part of the equator of the capsule of the lens, to which it is firmly attached in a tortuous line. Formerly, the suspensory ligament was described as being separated from the vitreous by a space—the canal of Petit—which was thought to be occupied by lymph during life. This canal is now generally considered to be the posterior part of the posterior chamber of the eye, whose boundaries are, anteriorly the iris, internally the lens capsule, externally the ciliary body, and posteriorly the hyaline membrane of the vitreous. The chamber is divided into intercommunicating parts by the fibres of the suspensory ligament. The relation of the suspensory ligament to the surrounding structures is of great practical importance, more especially with regard to accommodation, to the extraction of cataract, and to dislocation of the lens. The function of the suspensory ligament is probably that of maintaining the lens *in situ*, and of controlling its accommodative changes. It offers no obstacle to the interchange of fluids between the aqueous and vitreous chambers.

CONGENITAL DEFECTS.

Lenticonus, or conicity of the lens, occurs either at its anterior or posterior pole. It is recognised by a circular

shadow seen occupying the pupillary area on retinoscopy, by distortion of the retinal vessels on direct ophthalmoscopic examination, and by the centre of the lens being more highly refractive than its peripheral part. All these signs are also present in conical cornea, but, by the use of the keratoscope or Javal's ophthalmometer, the latter as the cause of these signs can be excluded.

Retinoscopy in an eye with lenticonus may give a very characteristic appearance. The rotation of a plane mirror causes a shadow which moves in the same direction as the mirror in the centre, but in the opposite direction at the periphery of the pupil. Opacities are common in lenticonus, and are probably congenital. Allied to lenticonus are lenses whose nuclei are eccentric, conicity being often combined with this condition.

Coloboma lentis is a congenital deficiency occasionally seen. The deficiency usually appears in the form of a small indentation in the lower and inner margin of the lens, or even as a mere flattening of the natural convex edge. It may, however, involve as much as a quarter of the substance of the lens. It frequently coexists with a congenital displacement of the lens, and in these cases the part of the lens which is ill developed is exactly opposite to the direction in which the lens is dislocated. A coloboma of the suspensory ligament is generally also present, and it is probable that this maldevelopment of the zonule is the cause of the coloboma of the lens (see Development of the Lens). Iridic and choroidal colobomata are often also present.

CATARACT.

Cataract is an opaque condition of the crystalline lens, which is due to structural changes of its component fibres. The opacity varies so much in the portion of the lens which is first affected, in its rate of progress, in the time of life at which it occurs, in its colour and consistency, and in its causes, that it is difficult to construct a good classification.

Varieties of Cataract.

The following arrangement of the different forms of cataract may be found useful: 1. Nuclear; 2. Cortical;

3. Lamellar ; 4. Pyramidal ; 5. Posterior polar ; 6. General or mixed.

1. Nuclear or Central Cataract.—In this form the opacity commences in the central portion of the lens, and gradually shades off towards the periphery (see figs. 9 and 10, opposite p. 366 ; also fig. 73). Its rate of increase varies considerably, the whole lens in some cases becoming opaque in the course of a few months, whilst in others the cortex may remain clear for years. Its colour is usually that of amber ; sometimes it is almost white, in other cases brown, and occasionally quite black. It mostly occurs after the age of fifty—very frequently from fifty to fifty-five. It may, however, come on at any age, or be present at birth.

2. Cortical cataract commences on both surfaces of the lens in the form of pyramidal streaks, having their bases at the equator of the lens, and their apices directed towards its antero-posterior axis (see figs. 7 and 8, opposite p. 366 ; also fig. 73) ; these are usually irregular in length and breadth. They are at first quite covered by the iris, and can then only be seen by dilating the pupil. After a time, however, they encroach upon the central portion of the lens, and can be seen within the normal pupillary area. These streaks finally become united into a mass of cortical opacity ; the central portion also becomes opaque, and the whole lens is thus rendered cataractous. This form of opacity is of frequent occurrence in old people, and is but rarely seen before the age of fifty. When progressive, as it usually is, cortical cataract shows great variation in its rate of increase.

3. Lamellar or Zonular Cataract.—In this form both the central and the peripheral portions are unaffected, but a shell-like layer of opacity exists between the centre and the surface of the lens (see figs. 5 and 6, opposite p. 366 ; also fig. 73). The exact position of this lamina is variable, but it is usually between the inner and outer fourths of the substance of the lens. Upon oblique focal illumination the cataract will be seen to be whiter at its periphery than at its centre, also upon ophthalmoscopic examination the peripheral portion of the opacity will appear denser ; this is just the reverse to the condition seen upon examining a nuclear opacity. It is

DESCRIPTION OF PLATE.

FIG. 1.—¹ Partial Dislocation of Lens (backwards, downwards, and outwards).

„ 2.—Dislocation of Lens (forwards and downwards).

„ 3.—Anterior Polar or Pyramidal Cataract.

„ 4.—Pyramidal Cataract.

„ 5.—Lamellar or Zonular Cataract.

„ 6.—Lamellar Cataract.

„ 7.—Immature Cortical Cataract.

„ 8.—Immature Cortical Cataract.

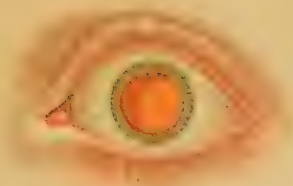
„ 9.—Immature Nuclear Cataract.

„ 10.—Immature Nuclear Cataract.

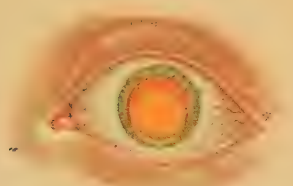
„ 11.—Posterior Polar Cataract.

„ 12.—Posterior Polar Cataract.

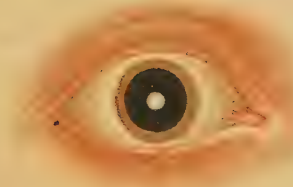
¹ N.B.—The figures in which the pupil is red represent the eye as seen by using the ophthalmoscope mirror ; others, as seen by the oblique focal illumination.



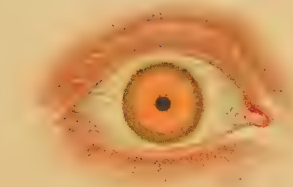
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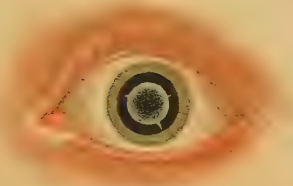
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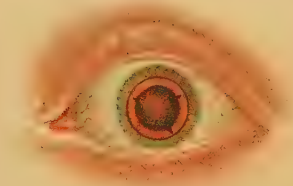
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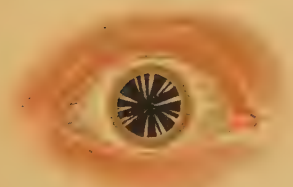
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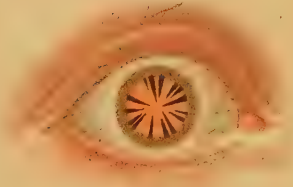
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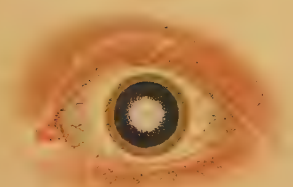
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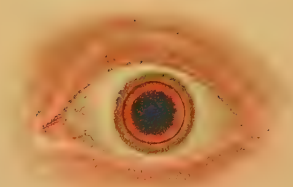
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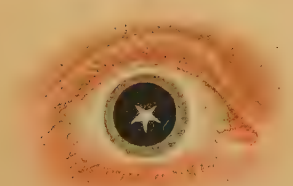
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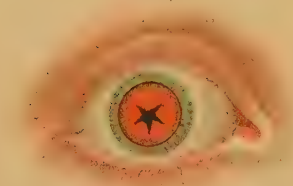
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generally very thin and delicate in structure, and has a faint bluish-white semi-transparent appearance; its surface is smooth, or only slightly granular; and, if this condition continues, it appears to remain stationary; occasionally, however, there appear dots of denser opacity upon its surface, which increase at the expense of the peripheral portion of the lens, and may often be seen as delicate radial projections directed towards the surface. The presence of these cortical opacities generally indicates that the cataract will progress, whilst their absence is in favour of a stationary condition. This is a point of great practical importance when the treatment of a lamellar cataract has to be considered.

Many points in the etiology of this form of lenticular opacities remain obscure. Since the growth of the lens takes place by means of new material formed on its surface, the occurrence of a cloudy layer of lens-substance at a certain depth would seem to indicate that, at some period of its growth, there had been interference with its nutrition which had led to the deposit, during that period, of imperfect lens material. Whether this period was before or after birth is disputed, but it appears that the average diameter of the opacity in zonular cataract is considerably less than the diameter of the lens at birth—a fact which points to the majority of these cataracts being prenatal in their origin.

Lamellar cataract is, almost without exception, bilateral, which points to some general malnutrition of the fœtus as the cause of the opacity. This is supported by the fact that in children with lamellar cataract a history of convulsions in infancy can generally be obtained, while there is usually a peculiar appearance of the permanent teeth, consisting in a defect in the enamel, which renders them of a bad colour; they as a rule present a constriction a little below the summit of the teeth, and the surface has a transversely corrugated appearance. These changes are usually most marked in the upper incisors. The exact relation between lamellar cataract, infantile convulsions, and defective enamel is somewhat doubtful. Rickets is considered by some to be the primary cause of these three conditions; but intra-uterine rachitis is very rare, and it seems more probable that where rickets is a concurrent

phenomenon it may be a product with the other three of a general epiblastic disturbance, resulting in a malnutrition, and so maldevelopment, of the epiblastic lens, the epiblastic enamel, and the epiblastic central nervous system.

Microscopically, a lens with zonular cataract is found to be minutely vacuolated. The extreme cortex is, as a rule, free from these vacuoles, which are very numerous in an intermediate zone, though the centre of the lens is never quite without them.

4. **Anterior polar or pyramidal cataract** consists in a dense chalky-white circular patch of opacity at the anterior pole of the lens, on and immediately beneath its capsule (see figs. 3 and 4, opposite p. 366). This must be distinguished from the condition where portions of the capsulo-pupillary membrane remain in front of the lens, and so produce an opacity, the lens, however, being clear, and therefore not cataractous. In anterior polar cataract the opacity is usually about 1 or 2 mm. in diameter, and when viewed from the side it is seen to stand out in front of the lens in a pyramidal form (fig. 73). Pathologically, the condition found varies with the age of the cataract. At first, proliferation of the subcapsular epithelial cells takes place, with a disintegration of the lenticular fibres in their immediate neighbourhood. Later, an ingrowth of cells from the margin of the opacity occurs, and so separates the structureless mass from the normal lens-matter. Subsequently, a hyaline membrane, identical in appearance with the capsule, is formed immediately superficial to this ingrowth, so that the cataract is completely enclosed by capsule, which appears to bifurcate at the margin. This new portion of the capsule seems to be the product of the ingrowth of the subcapsular epithelium, and favours the view that the normal lenticular capsule is epiblastic in origin.

The condition is sometimes congenital, the cause in these cases being probably a swelling of the cornea, the result of inflammation. This brings the anterior pole of the almost spherical lens in contact with the swollen and inflamed cornea, producing a localised cataract. Though perforation of the cornea does not seem to be necessary, still a perforating ulcer

following *ophthalmia neonatorum* is the commonest cause of pyramidal cataract; on the escape of the aqueous humour the lens has been pressed forwards against the cornea, the nutrition of the anterior pole being thus interfered with. In later life, a perforating ulcer may have the same effect.

Anterior polar cataract is best seen by oblique focal illumination. It is always stationary.

5. **Posterior polar cataract** is the term applied to any opacity situated on the posterior pole of the lens or its capsule. The opacity is usually small, round, and white; it not unfrequently has minute streaks radiating from it (see figs. 11 and 12, opposite p. 366). Posterior polar cataract may be congenital or acquired. The congenital form is

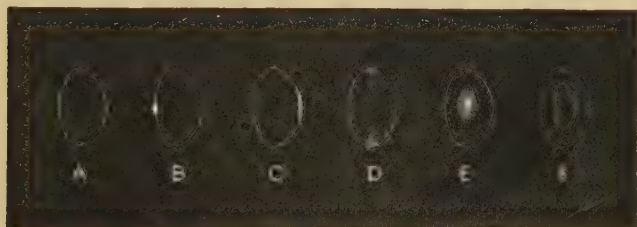


FIG. 73.—Cataractous Lenses, seen laterally.

probably in some way connected with imperfect absorption of the foetal hyaloid artery; and cases have been recorded in which a minute thread, corresponding in position and size to that structure, has been visible passing back from the opacity towards the optic disc. This is a spurious form of cataract, since there is no opacity of the lens or its capsule, but merely a small piece of the opaque capsulo-pupillary membrane adherent to the latter. The acquired form is generally progressive, and is nearly always secondary to pigmentary degeneration of the retina or chronic inflammatory changes in the choroid and ciliary body attended with numerous vitreous opacities. It is one of the later lesions found in progressive myopia. The change occurs in the posterior central radiating fibres of the lens (fig. 73).

6. **General or mixed cataracts** include all those in which the opacity occurs both in the cortex and nucleus, whether

these are completely opaque or merely dotted throughout with spots or striæ of opacity. Such cataracts are met with in endless variety, and no useful purpose would be served by a more detailed classification of them.

Many of the *congenital cataracts* would come under this heading. These may occur in one or both eyes. Usually the whole lens is opaque; but exceptional forms occur, such as the anterior and posterior polar, and cataracts in which the opacity is distributed irregularly. These congenital cataracts may be formed in one of two ways: either normal lens structure is laid down with subsequent disintegration, owing to intra-uterine inflammation; or the development of the lens is interfered with, opaque instead of transparent fibres being the result. Not unfrequently, in congenital cataracts the pupil acts very imperfectly to atropine, and the eye is often defective in other respects, so that, even after a successful operation, the vision is not good.

Cataracts are generally classed as being either *hard* (senile) or *soft*; and, although all intermediate degrees of consistency are met with, the distinction has a practical importance, as the two classes are amenable to different modes of treatment. The soft, if broken up, are readily dissolved by the aqueous, and can be absorbed with that fluid, while it is impossible to extract the lens from its capsule *en masse*. The hard cataracts, on the contrary, when broken up, imbibe the aqueous humour, and undergo much swelling, but show little tendency to become absorbed; on the other hand, when they have reached a certain stage of maturity, they can be shelled out entire from the capsule. One may say that, as a rule (to which there are many exceptions), cataracts which occur before the age of thirty or thirty-five are soft, and those occurring after that age are hard.

A hard cataract which has reached its full development may undergo pathological softening; this usually begins in the cortical portion of the lens, which becomes more or less milky in appearance. Sometimes the fluidity of the cortical structure is such that the harder central portion (nucleus) floats about; this constitutes what is known as the *cataract of Morgagni*.

Etiology and Pathology of Cataract.

The causes of cataract are still very obscure. The opacity appears to be due to an atrophic granular degeneration of the lens fibres. This is probably the result of defective nutrition, although it is frequently developed without any perceptible local or general cause.

1. **Senility.**—In many cases the disease appears to be due to the decline of vitality in the tissues of the body, either from age, anxiety, or dissipated habits. The so-called senile form of cataract may occur at any age after about thirty-five. It is thought by some that a shrinking nucleus produces cavities between it and the cortex, into which cavities fluid passes from the surrounding structures. Disintegration of the lenticular fibres is produced by the imbibition of this fluid. Others explain the production of the opacity by supposing that a block occurs at the exit of the stream of the nutritional fluid. There does not seem to be any very intimate connection between general arterio-sclerosis and cataract.

2. **Diabetes.**—A large proportion (about 6 per cent.) of diabetic patients suffer from cataract. This is as a rule of the soft variety, and matures slowly. The subject of a diabetic cataract is often young and usually under the age of forty. Both eyes are commonly affected. The exact pathology is uncertain. The lens probably becomes opaque on account of interference with its nutrition from the ciliary processes, due to the irritative action of the impure blood. Sugar is sometimes, but not necessarily, found in the opaque lenses; it has, however, been found in perfectly clear lenses. It is well to bear in mind that other ocular affections are common in this disease, such as *paresis of accommodation*, *amblyopia*, *hemianopsia*, *retinitis*, and *optic-nerve atrophy*. Nevertheless, diabetic cataracts may be operated upon successfully; in fact, many surgeons are of opinion that the eye recovers from the effect of the operation as readily as in health.

3. **Ergotism** has been observed to produce cataract; it is supposed to act by causing spasmodic contraction of the vessels of the ciliary body.

4. **Local diseases of the iris, choroid, or ciliary body, as**

in the secondary cataract of glaucoma, and of sympathetic disease.

5. **Injury.**—This may consist in a blow upon the globe, by which the capsule is ruptured—*concussion cataract*. The part of the capsule ruptured is usually in the neighbourhood of the equator. The lens is entirely or partially dislocated; it may be wounded by a sharp instrument, or a foreign body may have entered or passed through it. Opacity may follow a perforation caused by an ulcer of the cornea, as, for example, the pyramidal cataract.

6. **Convulsions.**—The possibility of convulsions being a cause of lamellar cataract has already been referred to (p. 367).

7. **Rare causes of cataract** have been found in cases of idiopathic tetanus, meningitis, malaria, syphilis, in the typhoid state, and during pregnancy and lactation. It is difficult to say how many of these may not be coincidental. An interesting form—*glass-blowers' cataract*—has recently been described. The posterior portions of the lens are usually first affected, the condition being slowly progressive. It frequently occurs under the age of forty.

An interesting fact that may be mentioned in this connection is that the average age for the onset of senile cataract in tropical climates is nearly twenty years earlier than in England.

The Symptoms and Diagnosis of Cataract.

The objective signs of cataract are best ascertained by the use of a convex lens and the ophthalmoscope. The examination is greatly facilitated by the previous dilatation of the pupil. Before using a mydriatic for this purpose, it is well to test the tension of the eye, lest the patient should be the subject of chronic glaucoma. One of the best mydriatics is a solution of cocaine and homatropine (F. 20). A few drops instilled into the palpebral sac at intervals of ten minutes will produce efficient dilatation of the pupil in the course of half an hour.

The patient should be examined in a dark room, and the light from a good ophthalmoscope lamp condensed upon the eye by means of a convex lens—oblique focal illumination

(see p. 110). Any opacity, either of the cornea or of the lens, will thus be brought into view, and will be seen in its normal colour of grey, amber, brown, or whatever it may be. We are thus able to localise the opacity, and so, to judge not only of the nature of the cataract but of the state of maturity to which it may have advanced.

In children, we notice whether there is a diffused haze over the whole lens substance, or whether there is a zone of opacity occupying its middle layers.

In adults, we notice whether the opacity seems to occupy the nuclear or the peripheral parts of the lens, or both of these parts together, or whether it consists of irregular dots or bars of opacity distributed irregularly over the lens. The denser the opacity and the more the substance of the lens affected, the less will be the darkness of the pupil, until, in a fully matured cataract, there will be scarcely any dark shadow of the iris visible in the pupillary area.

In thus examining the lens by focal illumination, the condition of the cornea should be carefully observed, as *nebulæ* and irregular surfaces which are difficult to see in diffused light are thus easily detected.

By direct ophthalmoscopic examination the condition of the lens can now be ascertained in another way. If the lens is quite clear there will be no impediment to the view of the vitreous and fundus beyond. If there is only slight opacity this will be rendered evident by turning on convex lenses—say +10 to +15, or +20 D—behind the mirror. Any opacity in the crystalline lens would intercept the rays reflected from the fundus, and so would appear dark (black) in proportion to its density. This is most useful in the very early stage of some cataracts, where the opacity is so slight that it may escape detection by focal illumination and yet may thus be seen as amorphous granules or vacuolated spaces.

The thickness of any opacity can in this way be approximately estimated by the amount of light which is reflected back from the fundus. In very slight and thin opacities we get a bright reflex, and can possibly examine the state of the fundus beyond. In denser opacities the brightness of the fundus reflex is less and less as the cataract becomes more mature, until it

is no longer visible. The characters presented by the various immature and partial cataracts when examined by these methods are given in the figures opposite p. 366 ; it will be observed that in the nuclear form the opacity is most dense at the centre, and gradually fades away at the outer part. When the cortical or central portion of the lens is sufficiently clear for an ophthalmoscopic examination to be made, advantage should be taken of this opportunity to ascertain the condition of the fundus ; such knowledge will be useful with regard to the probable results of a future operation, and cannot be obtained later when the cataract has become more general.

In the lamellar form, if the pupil is widely dilated, the periphery of the lens is seen to be clear, while in the centre of the pupil the shell of opacity forms a regular circular area of darker colour, which is often sufficiently thin to allow of the fundus being seen through it ; the edge of this often appears darker owing to the opaque shell being viewed 'end-on ;' occasionally minute striæ can be seen radiating from the opacity into the otherwise clear periphery.

The *catoptric test*, or *Sanson's images*, was formerly, before the introduction of the ophthalmoscope, a valuable method for proving the presence or absence of lenticular opacity. It is the test for demonstrating that, during the act of accommodation, the anterior surface of the lens bulges forwards to increase the refraction of the eye. If a light is thrown into the eye obliquely in a dark room, three reflection images are seen occupying the pupillary area, one from the cornea, a small, bright, and erect image ; one from the front of the lens, large, indistinct, and erect ; and a third reflex from the posterior surface of the lens, the smallest image, relatively brighter than the last but differing from the other two by being inverted, for it is reflected from the anterior surface of the vitreous, a concave mirror, whereas the other surfaces reflect as convex mirrors. If there is opacity of the lens, the inverted image cannot be seen. This test, in the present day, is called into use only to diagnose between hæmorrhage into the vitreous and a black cataract, for in both these affections the pupil may appear black by oblique focal illumination as well as upon ophthalmoscopic examination.

The subjective signs of cataract are chiefly troubles of vision which vary with the situation of the opacity. Central or nuclear opacities interfere more with the vision than those situated in the periphery of the lens. Partial opacities, situated in the path of rays entering the eye, give the sensation of fixed muscæ. These are rendered more evident by the patient looking through a narrow stenopaïc slit or a circular aperture. They are to be distinguished from the muscæ volitantes due to vitreous opacities by their coincidence with the movements of the eye; they do not continue to move after the eye has come to rest.

Elderly patients usually complain of failure of vision with rapidly increasing presbyopia, which has been unaccompanied by inflammatory symptoms. The failure is generally worse in one eye than in the other. Young subjects, on the other hand, seem to be near-sighted; that is, in reading, they will hold a book nearer to the eyes than normal so as to see the letters under a larger visual angle, and use more accommodative power at the same time. This necessitates greater convergence of the eyes, greater strain of accommodation, and a stooping posture, which together not unfrequently bring on real myopia, which at first was only apparent. The onset of myopia, varying in amount from 0.5 to 2 dioptries, the increase of pre-existing myopia, or the decrease of pre-existing hypermetropia in an elderly patient, must always be suggestive of incipient cataract, although no opacity of the lens can actually be seen by the methods just described. The increased refraction is due to the increase of the refractive index of the lens, and, in some cases, to the change of curvature of the surface of the lens on account of the swelling of its substance. Where astigmatism is found in such cases, the cornea will often be found to be free from astigmatism when examined with a Placido's keratoscope or with Javal's ophthalmometer.

With the progress of the cataract towards maturity, all useful vision disappears. First, all distant test types and objects are lost to sight; then the reading power, even for the largest type, gradually goes; lastly, the patient is unable to count fingers when held up within from 20 to 40 cm. of the affected eye. In no case of cataract, however, is the opacity

so dense as to prevent the patient from distinguishing between light and darkness. In the broad daylight, when placed with his face towards the window, he perceives a shadow when the hand is passed in front of the eyes—*perception of light*; and in a dark room he can localise the position of the flame of a lamp or candle—*projection of light*. *Perception of light* should always be present in mature cataract; its absence indicates the existence of disease in the fundus oculi; of course, in such a case no operation could be of any considerable benefit.

If there is perception of light but bad projection, a good result must not be expected by operation, though a certain amount of vision may be restored to the patient sufficient to elicit gratitude.

Owing to the changes which occur in the various sectors of the lens during the development of cataract, it sometimes happens that monocular diplopia and irregular astigmatism are developed.

Treatment of Cataract.

Many authentic cases have been recorded where a complete senile cataract has spontaneously disappeared. It is, however, extremely doubtful if the opacities in a senile cataract can become absorbed, leaving a clear lens; the lens is absorbed, with an aphakic result. Undoubted cases, however, of lenses with diabetic and traumatic opacities becoming again completely transparent have been recorded.

A hard cataract which has reached its full development may undergo pathological softening; this usually begins in the cortical portion of the lens, which becomes more or less milky in appearance. Sometimes the fluidity of the cortical structure is such that the harder central portion (the nucleus) floats about; this constitutes what is known as the *cataract of Morgagni*, and is probably the first stage in the occasional spontaneous absorption of a senile cataract.

It is extremely doubtful whether an opacity of the crystalline lens can be made to recede by the use of therapeutic agents, though it has been stated that improvement has been seen after an ointment of iodide of potassium has been rubbed

into the temples; the question of treatment therefore resolves itself into the best means of restoring vision by operative measures.

Before any operation is undertaken a careful examination of the patient must be made, special attention being paid to the following points:

(a) *The condition of eyelids, lachrymal apparatus, and conjunctiva.* Any blepharitis or conjunctivitis would court the onset of suppuration or inflammation as a complication of the operation. These diseases, if present, must therefore be first cured. For the same reason, lachrymal obstruction must be examined for; its presence is a strong contra-indication for any corneal section. Entropion, so common in the aged, would seriously interfere with healing of the corneal wound, and must therefore be treated first.

(b) *The condition of the iris.* A sluggish pupil or iridic adhesions suggest previous iritis, which may have been accompanied by choroiditis. Not only would the operation be rendered more difficult in its performance, but the prognosis would be more guarded.

(c) *The power of projecting light.* The examination of this must be conducted in a darkened room. Light is thrown, by means of a mirror, so that it falls on the various parts of the patient's retina, both central and peripheral, the subject being required to point to the direction from which the rays of light enter the eye. Failure in the central part of the field would make the surgeon very suspicious of senile choroidal atrophic changes. Peripheral limitation would point to diseases in the fundus oculi, such as retinal detachment, retinal hæmorrhage, and choroido-retinal atrophy. Vitreous opacities may exclude all perception of light. Diminution in the power of projecting light is not necessarily a contra-indication for operation, but the prognosis would not be so good.

(d) *The tension of the globe.* A diminished tension usually indicates vitreous disease or degeneration. Should operation be attempted with this condition, especial care must be taken to guard against prolapse of the vitreous.

(e) *The condition of the other eye.* It often happens that though one lens is completely cataractous, the other cataract

is only in an incipient stage, and the details of the fundus can be seen. Should this be so, a careful search for any pathological change must be made, as diseases of the fundus oculi are frequently bilateral.

(f) *The condition of the urine.* This is undertaken to ascertain whether the patient has any renal disease, or diabetes. Each may be accompanied by retinitis, and each may seriously interfere with the healing of the corneal wound, and introduce several complications, immediate or remote.

The treatment of cataract by operation may be divided into the following: 1. By artificial pupil; 2. By solution and absorption; and 3. By the extraction of the entire cataract.

1. By Artificial Pupil.—When the cataract is non-progressive, and the extent of the opacity is such that its area is equal to, or very slightly greater than, that of the normal pupil, much benefit is sometimes derived from the formation of an artificial pupil. In such cases the patient may be able to see tolerably well in the twilight with deeply tinted glasses, by shading the eyes, or by other conditions which favour the dilatation of the pupil; but is quite incapacitated for useful vision by the presence of diffused bright light, which causes contraction of the pupil. In order to ascertain the probable result of an artificial pupil in a case of this description, the pupil should be thoroughly dilated with atropine; the vision for distant types should then be carefully tested, any existing error of refraction being at the same time neutralised by means of the correcting glasses. If this dilatation of the pupil is found to materially improve the distant vision, so that the patient is enabled to see the letters corresponding to $\frac{6}{12}$, $\frac{6}{18}$, or even $\frac{6}{24}$ of Snellen, it may be anticipated that the vision will be still more improved by the formation of a small artificial pupil in the downward and inward direction; and that after the operation, when the accommodating power of the eye is no longer paralysed by atropine, he will also possess good near vision. The size of the artificial pupil must vary according to the extent of the opacity; so long as it is brought opposite to the clear portion of the lens, the smaller it is, the better will it be for distinctness of vision. It may be made by iridectomy, by iridotomy, or by iridodesis. The method I prefer in these cases is that of iridectomy by means of the

hook, as described on p. 234. The artificial pupil made in this manner is narrow, especially at its periphery, and there is not so much spherical aberration as occurs in larger iridec-tomies in which a considerable extent of the lens margin is exposed.

In suitable cases this operation possesses at least two advantages over the removal of the lens—namely, that the operation itself is practically free from risk ; and, secondly, that the power of accommodation is retained. It is especially indicated in polar cataracts, and in the non-progressive form of lamellar cataract.

When the distant vision is not improved by full dilatation of the pupil, it may be concluded that an artificial pupil would be of no service, and recourse had better be had to one of the operations to be presently described for the absorption or the removal of the lens itself.

2. By Solution and Absorption.—Any kind of cataract, whether nuclear, lamellar, cortical, or general, occurring in subjects under thirty-five years of age, is, as we have mentioned, *soft* in structure. By lacerating the anterior capsule, and breaking up the laminæ of such a lens, the aqueous humour is brought into immediate contact with its fibres, and has the effect of causing them to become opaque and swollen. This effect is produced within the first twenty-four hours after the operation, and is immediately followed by a process of gradual disintegration, solution, and absorption. This method is called *discission*, or the *needle operation*. It may be employed in any soft cataract which is not amenable to treatment by the formation of an artificial pupil. The younger the subject the more quickly do solution and absorption take place, and the less liable is the eye to severe inflammation after the operation. After the age of thirty the nuclear portion of the lens is so hard that the number of operations, and the time required for solution, are beyond endurance, while the larger size of the lens, and the greater intolerance of the eye to increased intra-ocular tension, render this operation more dangerous than in younger subjects.

The *needle operation* (*Discission*, *Solution*) gives so little pain that, except in young children and in persons of nervous temperament, general anæsthesia is not necessary. The pupil must be dilated by the previous use of a mydriatic (F.10, 11, 19,

20). The positions of the operator and the patient are the same as for iridectomy (p. 231). The lids being separated by a speculum, and the globe held steady by fixation forceps or with the fingers, a cataract needle (fig. 74) is passed obliquely through the outer part of the cornea into the anterior chamber. Its point is then made to perforate the anterior capsule of the lens within the area of the dilated pupil (see fig. 75). By gentle to-and-fro movements the capsule is now lacerated; and the lens matter having been broken up to the extent desired, the needle is gradually withdrawn. The best part of the cornea at which to insert the needle is that at from 2 or 3 mm. from the



FULL SIZE
FIG. 74.
Cataract
Needle.

outer extremity of its horizontal diameter. The extent to which the capsule should be lacerated, and the lens matter stirred up, depends upon the nature of the case. Care should be taken not to wound the posterior capsule of the lens, as the vitreous is then liable to come forward, and so to interfere with the action of the aqueous upon the lens. To prevent this accident needles are often made with a shoulder or 'stop;' this, however, is not a sufficient safeguard for a clumsy operator, and is quite unnecessary for any one of average dexterity; it has, nevertheless, a slight advantage, as it gives firmness to the needle. In a properly constructed cataract needle the shaft should exactly fit the puncture: if it fits too loosely aqueous will leak out, if too tightly its movements will be impeded. The complete solution of a lens by this

process usually requires the performance of three or four needle operations, and occupies a period varying from four to eight weeks. At the first needling it is best not to do more than lacerate the capsule and the most anterior layers of the lens substance by a slight vertical or crucial incision. This is usually followed by increased opacity of the lens substance, which swells up and bulges forwards through the pupil, so that it may be seen projecting into the anterior chamber. After the operation the pupil must be kept dilated by the use of 0.5 per cent. solution of atropine three times a day; the patient should be kept in bed, the room darkened,

and the lids kept closed by means of sterilised gamgee tissue and a bandage or adhesive plaster ; both the eyes should be screened from the light, either by means of a dark shade over the bandage, or by remaining in the dark room.

Complications.—Although a simple and easy operation, several precautions are necessary.

(i) The laceration of the capsule and the lens must not be too extensive, especially at the first needling, otherwise the masses of crystalline lens become so rapidly swollen by imbibition of the aqueous as to set up increased intra-ocular tension. For similar reasons the iris and ciliary body are liable to

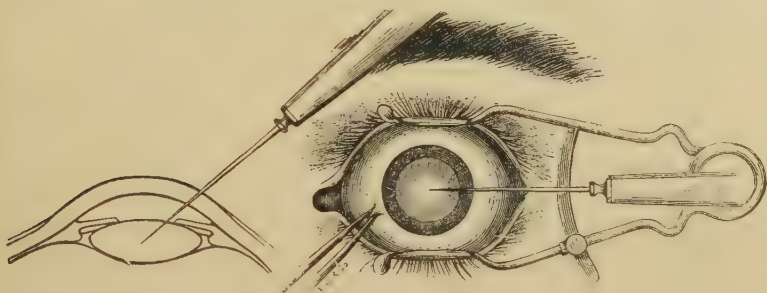


FIG. 75.—The Needle Operation.

become irritated by the swollen lens to such an extent as to cause iritis or irido-cyclitis.

(ii) During the three days succeeding the operation the eye requires careful watching and treatment. The occurrence of slight ciliary congestion, without pain, need cause no anxiety ; but if the redness around the circumference of the cornea increase, and be accompanied by pain, and by symptoms of commencing iritis, the atropine should be repeated more frequently, and boracic fomentations applied. If these remedies do not cut short the inflammatory symptoms, but are followed by increasing pain, congestion, and symptoms of irido-cyclitis, or glaucomatous tension, the soft lens matter must be immediately removed, either by the method of linear extraction or by suction.

The exact period at which to perform the *second needling* must be decided by the condition of the eye. In no case should

it be undertaken until all the inflammatory symptoms which may have been produced by the first operation have entirely subsided, leaving the eye perfectly quiet, free from all pain, and without a trace of redness in the circumcorneal zone. As a rule, it is well to wait until the process of absorption seems to be at a standstill; if, however, it is wished to hasten the process, there is no objection to repeating the needling as soon as all irritation has ceased.

In the second and third operations the needle may be used more freely than in the first, as there is less risk of setting up inflammatory mischief. When absorption progresses slowly, some surgeons perform repeated paracenteses of the anterior chamber in order to evacuate the aqueous humour, which is saturated with the substance of the lens.

The needle operation is often required after the extraction of cataract, when a portion of so-called opaque capsule lies in the pupil (see p. 408).

The needle operation is often supplemented by the subsequent removal of the soft lens matter, either by linear extraction or by suction. These operations save a good deal of time, and are sometimes necessary, as we have seen, to counteract inflammatory symptoms after a simple needling.

The *linear operation* (Gibson) consists in the removal of a soft lens through a small incision in the cornea. It is especially indicated in cases of traumatic cataract, whether produced by the needle operation or by any other injury, in which the eye has become painful and inflamed. When employed for the removal of other forms of soft partial cataract, as the lamellar, the linear extraction should be preceded by the needle operation (p. 379), the anterior capsule of the lens being *freely* lacerated, in order that the lens matter may be rendered more soft and so escape more freely from the corneal wound. Some surgeons, however, prefer to complete the operation at one sitting, and in order to do this they lacerate the anterior capsule of the lens by means of a cystitome (fig. 91) introduced on the flat, through the corneal wound.

When the needle operation is performed as the first stage of the operation, the interval of time which should elapse

between this and the extraction of the softened lens matter must vary with the condition of the eye. Should the latter remain quiet, and free from any marked pain or redness, it may with advantage be left until the sixth or eighth day. But should there be considerable pain, and especially if this is combined with inflammatory or glaucomatous symptoms, the extraction should be effected without further delay.

The pupil being widely dilated by atropine, the eye thoroughly anæsthetised by cocaine, the eyelids separated by a speculum, and the globe held steady by fixation forceps, a bent broad needle (fig. 56) is passed through the cornea into the anterior chamber in a direction parallel to the plane of the iris. The incision should be about 2 mm. within the margin of the cornea near the upper periphery; its width should be about 5 or 6 mm. If the greater part of the lens substance still lies within the capsule, the latter should be freely incised before the needle is withdrawn. The broad needle is then withdrawn and laid aside, and the curette (fig. 76) taken up. Gentle pressure is first made with this upon the upper lip of the wound, and is usually followed by the exit of a considerable quantity of aqueous humour and soft lens matter; the curette may then be carefully introduced through the wound into the area of the pupil, when any remaining lens matter will usually be found to escape along its groove. Should any fragments of lens still remain, they may be evacuated through the wound by gently stroking the cornea from below upwards with the back of the curette.



FIG. 76.
Curette.

Accidents and complications.—(i) Care must be taken in using the curette not to rupture the posterior capsule; this accident is liable to be followed by protrusion of the vitreous forwards into the anterior chamber and through the corneal wound. If this should occur, no further attempt should be made to remove the lens matter.

(ii) Unless the curette be gently manipulated, the iris may be contused; a slight injury of this structure is liable to be followed by inflammation and plastic exudation.

(iii) At the time of the first escape of the aqueous, after the incision of the cornea, the iris is occasionally found to protrude between the lips of the wound. This can often be returned by gentle pressure with the curette or spatula. Should it be found impossible to do this, the protruding portion must be seized with forceps and excised with the iridectomy scissors. Some surgeons prefer to remove a small piece of iris in all cases.

The after-treatment is the same as for the needle operation.

The *suction operation* is similar in principle to that just described, and, like it, may be performed all at one sitting, but is generally more successful when preceded by the needle operation. It consists in the removal of the soft lens matter by means of an aspirator passed through a small wound in the cornea. The same interval of time, &c., between the needling and the removal of lens matter is necessary here as in the linear operation.

The eye being cocaineised and fixed as before, an incision is made in the cornea by means of an angular broad needle (fig. 56, p. 233); the wound should be just large enough to easily admit the nozzle of the aspirator; it should be on the temporal side of the cornea, near the sclero-corneal junction. The aspirator consists of a small flattened cannula, having a free opening on one side (*b*, fig. 77), and connected with a glass tube (*d d*). This is attached either to a metal piston-syringe (Bowman) or to an indiarubber tube and mouthpiece (*e*) (Teale). The nozzle of this instrument is passed into the anterior chamber with its concavity upwards, and placed in the most favourable position for withdrawing the lens matter without injuring the iris. It is well to keep the nozzle of the syringe well in view just behind the cornea in the area of the pupil; it should not be allowed to dip deeply behind the plane of the iris in search of fragments, lest it tap the vitreous instead of the lens matter. Gentle suction is then made, and as much lens matter removed as possible.

The after-treatment is the same as for the needle and the linear operations.

This operation requires great care and delicacy in manipulation; when successful it gives very satisfactory results, more

especially in the saving of time which it effects by the early removal of the lens matter.

Strict antiseptic precautions, especially with regard to the nozzle of the instrument, should be exercised here as in all cataract operations. Unfortunately it is occasionally followed by inflammatory trouble, which sometimes leads to loss of the eye by suppuration. The first symptom of such a mishap is a continuance of the conjunctival injection and pain beyond the third day; signs of iritis then supervene—dulness of the iris and incomplete dilatation with atropine; a day or two later hypopyon may make its appearance. A good-sized iridectomy downwards will sometimes do good in this state of affairs, and occasionally the pus will be absorbed and a good

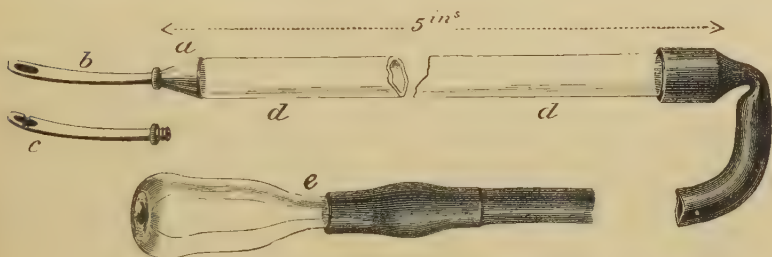


FIG. 77.—Teale's Suction Apparatus for Cataract.

result obtained; the suppuration may, however, extend to the vitreous, and shrinking of the globe ensue.

3. By the **Extraction of the Entire Cataract.**—After the age of thirty the structure of the lens is so dense, and its nucleus so large, that its removal requires a larger incision than that just mentioned for the linear operation.

It is usually advisable to avoid operating until the opacity has extended to the greater part of the lens, otherwise the unaffected cortical substance remains adherent to the capsule; and although, owing to its transparency, it is difficult or impossible to see it at the time of the operation, it afterwards becomes swollen and opaque, and gives rise to trouble to be presently described. When the whole of the lens has become opaque, the cataract is said to be 'mature' or 'ripe;' the signs of this condition are, that no red reflex can be obtained from the choroid

by the use of the ophthalmoscope, and no shadow is thrown by the iris upon the lens when light is projected upon the eye by oblique focal illumination. If the cataract is removed before it has reached this condition of maturity, it does not so readily shell out from the capsule, but is liable to leave behind it more or less of the transparent portion either adherent to the capsule or within the pupillary area. These remains, however, can often be evacuated at the time of the operation; when left in the eye they are apt to swell up and to cause iritis. In such cases lymph is often thrown out in considerable quantity, and, becoming organised, may form a dense membrane completely occluding the pupil. As a rule, these fragments of cortical matter are eventually absorbed, but in the meantime irreparable mischief may have been set up by their presence.

There are, however, many circumstances which sometimes render it highly inconvenient, if not altogether impossible, to wait for the complete maturity of a cataract. There may be commencing, or equally advanced, cataract in the second eye, by which the patient is deprived of all useful vision, and is consequently debarred from following his usual occupation. The patient's place of residence may be beyond the reach of surgical skill, and he may be unable to present himself for periodical examination. In such cases the extraction of the immature cataract at the earliest possible date is imperative. Under such circumstances it is best to perform an iridectomy upwards as a preliminary operation, and after the lapse of six or eight weeks to extract the cataract from one eye at a time. This *preliminary iridectomy* does not interfere with what little vision the patient may possess—indeed, the enlargement of the pupil may improve this, and it has the effect of lessening the danger of iritis after the extraction; it also enables the cataract to be removed before it is quite mature without much risk, and in some cases seems to hasten the maturing of the cataract.

When one eye only is affected, or when the second eye is still serviceable, the removal of the lens is less urgent; if, however, the cataract is quite complete, it is better that it should be extracted without further delay. The result of the operation will not be so satisfactory to the patient as it would be if the second eye were blind, on account of the difference of refraction between

the operated and the sound eye ; but delay in extraction might cause the cataractous lens to undergo fatty degeneration and to set up inflammatory trouble in the eye, and so prevent the possibility of a successful operation. The increased visual field which is obtained by the use of both eyes is of considerable advantage, while the operated eye will be ready for use in case of the other becoming cataractous. Finally, the removal of a disfigurement, which is often very marked, is of importance from an æsthetic point of view.

When both eyes are affected at the same time, and both the cataracts mature, it is well that the two extractions should not be performed at the same sitting, but that they should be separated by an interval of some weeks. If both eyes were done together, and one of them should progress badly, it would complicate the management of its fellow ; whilst in two separate operations, the failure of the first eye, during or after extraction, may enable us to take special precautions with the second ; thus it might be considered better to make the incision more or less peripheral, to perform preliminary iridectomy, to extract the lens in its capsule, or to use the scoop instead of pressing upon the cornea in the removal of the lens.

Artificial maturation of immature cataracts is practised by some surgeons in order to accelerate the restoration of vision by means of operation. Various methods of producing the artificial maturation are recommended ; they are chiefly modifications of the method of Förster.¹

Förster's method of artificial maturation consists in performing a preliminary iridectomy. This in itself produces a disturbance in the clear cortical matter by the lens starting forwards at the escape of the aqueous humour ; he increases this disturbance by gently rubbing the cornea with the blunt angle of a tenotomy hook, or with the closed iris forceps, immediately after the iridectomy. The increase in the opacity caused by this treatment may be so great as to shut off all choroidal reflex in six days. The lens may be extracted about one or two months after this ripening process. Iritis may follow ; and if the manipulation has been too severe, vitreous will almost certainly escape at the time of the extraction.

¹ *Archives of Ophthalmology*, vol. xi. (1882), p. 344.

Nuclear cataracts are preferable to cortical in choosing a case for this treatment.

Theoretically, of course, this operation is delightful. Its advocates maintain that it shortens the period of useless vision, restores useful sight within a definite time, and is attended with little or no risk of destruction to the eye. Practically, however, and judging from what I have seen in the practice of others, I am of opinion that it would be safer to extract an immature lens than to injure it by this method of procedure, which can hardly fail to set up inflammation in the ciliary body, iris, cornea, and other parts.

Other methods of artificially maturing cataract have been practised, such as paracentesis followed by external massage, and a preliminary needling. Neither of these is free from risks.

The flap operation.—It was not until towards the middle of the eighteenth century that the operation of extracting a cataractous lens became a regular surgical proceeding. Previous to that date, the recognised treatment of cataract was that of *reclination* or *couching*, which consisted in dislocating the lens into the vitreous. The immediate effect of this was of course satisfactory as regards the improvement in vision, but subsequent trouble nearly always arose from the irritation set up by the displaced lens, and the eye was generally eventually lost from irido-choroiditis or glaucoma.

During the first half of the eighteenth century extraction was occasionally performed; but to Daviel certainly belongs the credit of having definitely established the superiority of extraction over reclination.



FIG. 78.
Daviel's
Incision.

Daviel's method was to make in the cornea near its lower margin an incision with a lance-shaped knife, and to enlarge this in both directions with scissors, so that he obtained an incision concentric with the lower margin of the cornea, and extending a little above the horizontal meridian (fig. 78).

The flap having been raised, the capsule was lacerated, and the lens expressed through the pupil.

Beer modified the operation by using the triangular knife which bears his name. The point of this was introduced into the cornea level with its horizontal meridian, and, while the

point of the knife was carried across the anterior chamber to make its exit at a corresponding point on the other side, the edge cut its way out at the sclero-corneal junction, thus forming a flap which corresponded almost exactly with the lower half of the cornea (fig. 80). The flap thus formed was slightly smaller

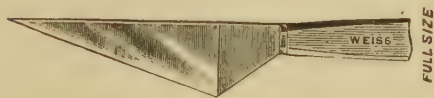


FIG. 79.—Beer's Knife.



FIG. 80.—Beer's Incision.

than Daviel's, and, being made by a simple cut, allowed of more perfect adaptation of the parts.

There is no doubt that the above method was a very great advance on former proceedings, and that many most excellent results were obtained by it; indeed, nothing could be more perfect than a flap operation which succeeded well: after the wound had healed there was hardly a trace of its existence left upon the cornea, and the pupil retained its natural size, form, and function; but the percentage of failure was very high, and this was in great part due to faults inherent in the method.

In the first place, the nutrition of the cornea was seriously imperilled by a section including half its circumference; in the second, the large size of the wound predisposed to prolapse of the iris, which not only delayed the union of the wound, but by its adhesion gave rise to subsequent inflammatory trouble, such as iritis and irido-cyclitis. The iris itself was, moreover, necessarily contused by the passage of the lens through the pupil, and this was often followed by iritis, which led to closure of the pupil by lymph.

Some of these dangers were lessened by *Jacobson*, who made the section in the sclerotic concentric with the cornea, thus carrying it through vascular tissue, while, owing to the larger circumference of the globe here, the same length of incision was obtained without carrying its extremities as high as the horizontal meridian (fig. 81). It is doubtful whether to *Mooren* or to *Jacobson* should be ascribed the credit of adding an iridectomy, thus obviating effects of contusion of the iris and preventing its prolapse.



FIG. 81.
Jacobson's
Incision.

Undoubtedly the most important modification since the introduction of the operation of extraction is that which constitutes von Graefe's operation. The principle of his operation is, that the section should be as near an approach to a straight line as possible, since a linear wound allows of a more perfect coaptation than a flap; that the wound should by preference be entirely in the sclerotic; and that an iridectomy should form part of the operation.

Since the wound must have a minimum extent of 10 mm., and the ciliary body must be avoided, the direction of a 'linear' section which is to be wholly in the sclerotic allows of comparatively little variation.

Von Graefe's Linear Operation.—This, with slight modifications, is the form of operation that is almost universally performed at the present time for the extraction of a hard cataract.

1. *Preliminary measures.*—Great care must be taken with the general condition of the patient. Any cough or cold must be first cured, as an attack of coughing or sneezing, either during the operation or immediately after, might spoil all. The bowels should be thoroughly emptied previous to the operation, in order that the patient may be kept perfectly quiet for twenty-four hours after the cataract has been extracted. It is very important to gain the full confidence of the patient, as great help may be afforded the operator. For ten minutes before the operation both eyes must be thoroughly cocainised. This may be secured by using a previously sterilised 4 per cent. solution of the hydrochlorate of cocaine. Care must be taken to keep the eye closed while this is being done, to prevent any corneal desiccation which is otherwise liable to take place. Simultaneously with the use of the cocaine, a 2 per cent. solution of eserine should be instilled into the eye that is to be operated upon. This renders the performance of an iridectomy easier, besides diminishing the risk of prolapse of the iris.

Immediately before the operation is begun the eyelids must be washed externally and internally with an antiseptic lotion (F. 30, 31), to be followed by irrigation of the palpebral aperture by sterilised normal saline solution. The instruments

must be boiled in a solution of bicarbonate of soda, and laid by the side of the surgeon in the order in which they will be used. A skilled assistant should be in attendance.

It is then very important that the operator should give instructions to the patient. The other eye should be kept open, and his attention fixed as the surgeon directs. The mouth and hands are to be opened; on no account must the patient hold the nurse's hand. He must be told that there will be slight pain, and on no account squeeze the eyes; instead he must breathe deeply.

2. The *incision* is made with the right hand for the right eye, and the left hand for the left eye, the surgeon standing behind the patient's head.

The eyelids are kept open by means of a stop speculum. There are several varieties of this instrument. The form I prefer for this operation is that shown in fig. 82. It is curved

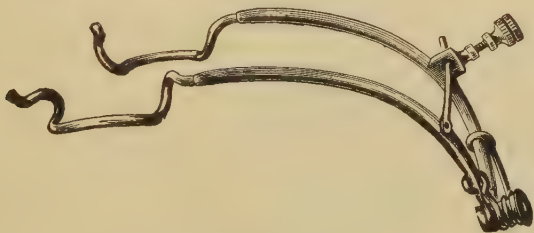


FIG. 82.—Spring Stop Speculum.

in such a manner as not to impede the movement of the instruments used, and its outer end, being well behind the plane of the eye, can, if necessary, be held by an assistant without interfering with the operator.

Noyes's specula (fig. 83) are also admirably adapted for cataract extraction.

Still better than the speculum is the separation of the lids by the fingers of a good assistant. The speculum undoubtedly holds the lids well apart, but it also affords a fulcrum by which, if the patient should make a violent attempt to close the lids, the vitreous is more likely to be extruded by the *vis a tergo*.

Anderson Critchett¹ prefers to elevate the upper lid by means of his own finger, which he does with the third finger

¹ *Trans. Soc. Française d'Ophthalmologie*, 1886, p. 320.

of the hand holding the fixation forceps as shown in fig. 84. He claims for this method that he can raise the lid sufficiently

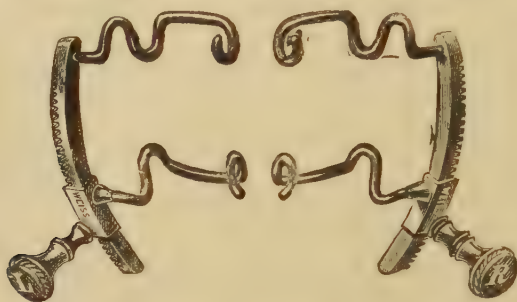


FIG. 83.—Noyes's Specula (right and left).

for the incision without causing discomfort to the patient, and that in the event of any involuntary spasmodic contraction on

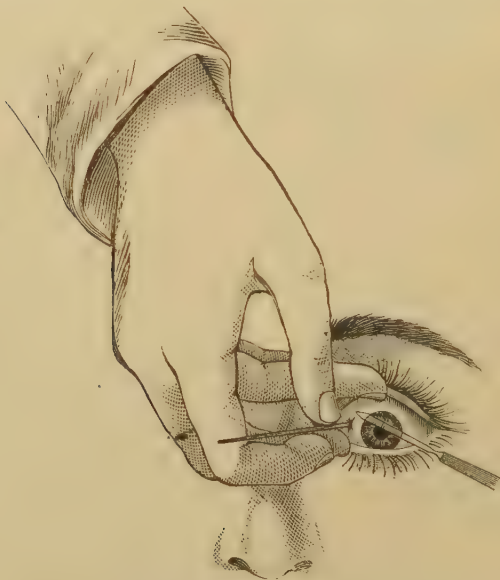
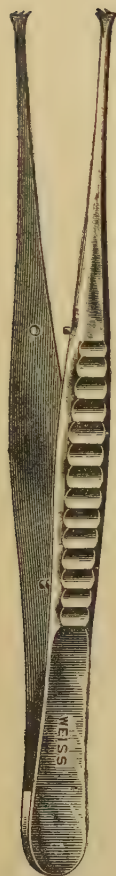


FIG. 84.—Nature's Speculum (Anderson Critchett).

the part of the patient, he can relax the lid immediately and so prevent the calamity of losing the vitreous humour.

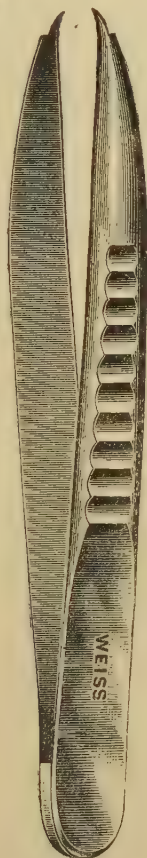
The globe must be held steady, and kept under the control

of the operator by some fixation instrument. The conjunctiva may be seized just below the position of the counter-puncture



FULL SIZE

FIG. 85.—Fixation Forceps.



FULL SIZE

FIG. 86.—Rat-toothed Fixation Forceps.



FIG. 87.—Von Graefe's Linear Cataract Knife.

with the fixation forceps (fig. 85), or when the conjunctiva is extremely brittle the sclerotic may be held by means of a forceps with sharper and longer teeth (fig. 86).

Von Graefe's linear knife (fig. 87), held with its cutting edge upwards, is then made to enter the sclerotic at a point 3 mm. below the upper tangent of the vertical meridian, and lying on the tangent of the transverse meridian of the cornea (A, fig. 88), and to penetrate the anterior chamber; the direction of this penetration should be downwards and inwards towards c (fig. 88); the knife having reached the middle of the anterior chamber, its handle is slightly depressed, and its point pushed steadily onwards in front of the plane of the iris, so that a counter-puncture may be made in the sclerotic on the opposite side, in a position which should correspond to that of the puncture (B, fig. 88). The knife is now made to cut its way upwards through the sclerotic, and to come out at the junction of this with the upper part of the cornea; this is effected by pushing the knife steadily onwards as far as its heel, and then withdrawing it if necessary.



FIG. 88.
Von Graefe's
Incision.

The above incision, which ordinarily goes by the name *von Graefe's*, has been slightly modified by different operators, and von Graefe himself at one time made the puncture and counter-puncture somewhat higher, so that the height of the flap was less than a millimetre.¹ In nearly all modern operations the puncture and counter-puncture are made a little beyond the sclero-corneal junction, and from 2 to 4 mm. below the horizontal tangent of the cornea; the line of incision in some instances traverses the cornea, in others the sclero-corneal junction or the sclerotic.

Fig. 89 represents the modification of this incision which, in suitable cases, I usually adopt in my own practice. The puncture and counter-puncture are made in the sclero-corneal junction and 3 mm. below its upper tangent; the knife is brought out through the sclero-corneal junction above—as represented by the dotted line.

It has been found that if a short conjunctival flap be made to cover the corneal wound, healing is accelerated. The flap should be 2 or 3 mm. in length, and during the performance of the iridectomy and delivery of the lens, must be turned forwards on to the cornea, otherwise it would interfere with those stages of the operation. At the end of the operation, it must again be turned upwards over the wound.

¹ Vide letter of von Graefe in de Wecker's *Chirurgie Oculaire*, p. 30. Paris, 1879.

3. *The iridectomy.*—The fixation forceps are now entrusted to the assistant, who, if necessary, will seize the ocular conjunctiva below the cornea, and gently rotate the globe downwards. The iris is now to be seized with the iris forceps near its pupillary edge, and drawn just outside one angle of the wound; whilst slight traction is made upon it in this position, its outer part is gripped through with the iris scissors in

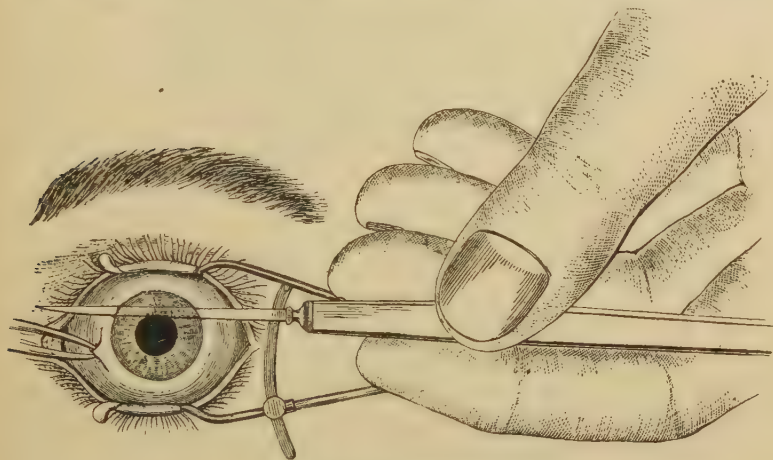


FIG. 89.—The Incision.

the manner shown in fig. 90; the portion of the iris held in the forceps is then gently drawn across to the other angle, and the excision completed as near to the periphery as possible.

There has been considerable controversy of late years concerning the performance of iridectomy in the operation of cataract extraction. There are many surgeons who consider iridectomy an unnecessary mutilation of the eye, an operation more liable to set up iritis, and attended with optical defects, besides being a permanent disfigurement. On the other hand, there are powerful advocates for the performance of iridectomy, and these hold to the opinion that cataract extraction with iridectomy is an operation less likely to be attended with prolapse of the iris, that the delivery of the lens is brought about with less force, so that the iris is not subjected to

bruising, and the fear of subsequent destructive irido-cyclitis is reduced to a minimum.

This is not the place to discuss the points in favour of or

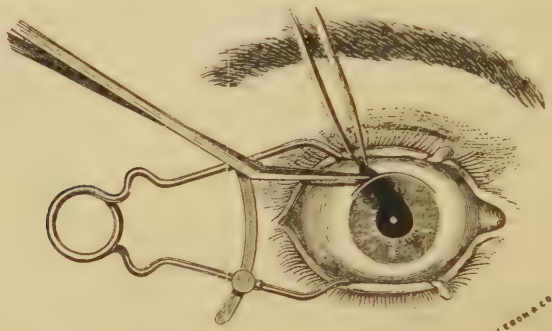


FIG. 90.—The Iridectomy.

against such a proceeding, beyond mentioning that the optical results *without iridectomy* are decidedly better should the operation be successful; but the liability to anterior synechiæ, prolapse of iris, and involvement of iris in the cicatrix with irido-cyclitis, is sufficient to raise doubts as to which is the better procedure.



FULL SIZE

FIG. 91.

Cystitome
and Curette.

4. The laceration of the anterior capsule of the lens is the next step in the operation. The operator again takes the fixation forceps in order to steady the globe with his left hand. The cystitome (fig. 91) is now to be gently passed, on the flat, into the anterior chamber; when it has reached the lower edge of the pupil its point is rotated towards the capsule, and the latter is freely lacerated from below upwards, and from side to side. In doing this it should be remembered that the capsule tears very readily, and that any undue pressure on the lens may cause it to sink back into the vitreous. The elasticity of the capsule causes the rent made by the cystitome to gape widely, so that

if properly incised a large triangular gap is left after the lens has been removed.

Some operators prefer to use a pair of toothed forceps, somewhat larger than iris forceps, instead of the cystitome; with this the central portion of the anterior capsule is seized and torn out. Several varieties of this kind of instrument are made by Weiss.

5. *The removal of the lens.*—In order to bring about the delivery of the lens, the eye must be gently rotated downwards by means of the fixation forceps; it will also greatly assist the operator if the patient will endeavour to look downwards at the same time. Then by the exercise of gentle pressure

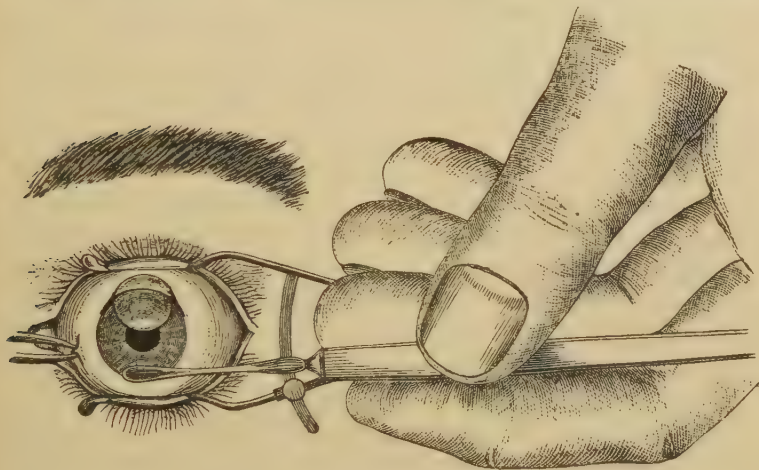


FIG. 92.—The Removal of the Lens.

with the back of the curette upon the sclerotic and the lower part of the cornea, the edges of the wound are seen to become separated, and the upper edge of the lens presents itself between them; by the continuation of this pressure in a direction backwards, and slightly upwards, the lens is presently expelled (fig. 92). In immature cataracts there will still remain a certain amount of soft cortical matter within the anterior chamber. This should, as far as possible, be evacuated at once. Its removal may be attempted before the speculum is taken out, by gently stroking the cornea with the back of the curette from below upwards towards the wound;

or, the speculum being removed, a similar pressure may be made upon the cornea through the lower lid, either with the finger or the back of the curette. Either one or other of these methods of coaxing out the soft matter should be repeated until the pupil looks quite black and clear.

6. Finally, the condition of the edges of the wound has to be looked after. It is a good plan to pass the iris forceps between the lips of the wound and gently grope for any tags of transparent capsule that may be present; if such be found they should be snipped off with the scissors. If any portion of the iris is prolapsed into or through the wound it must be similarly treated. The curette or tortoiseshell spatula should then be passed just within the wound, so as to push the iris into its proper position, and to procure an exact adaptation of its flaps.

Operation without Iridectomy.—The incision is made in the same way as in the former operation. The capsule of the lens is now lacerated through the pupil, and pressure is made below at the sclero-corneal junction, so as to tilt the upper margin of the lens forwards and to cause the cataract to present through the wound; as it does so it pushes the iris out with it, which either retracts by itself or has to be carefully replaced with the curette.

Irrigation of the Anterior Chamber.—Should lens matter still remain after the careful *nettoyage* just described, it is well to have recourse to irrigation. This method was introduced some years ago by M'Keown, who uses a glass syringe with a flat nozzle. Wickerkiewitz of Posen has constructed an elegant glass bottle provided with a platinum nozzle, in which water can be sterilised by boiling and then allowed to cool to 100° Fahr. before using. It is called 'Undina' from its shape, which is similar to that of Undina's lamp. It is made by Luer of Paris.

I have of late adopted a similar method for getting rid of soft lens matter from the anterior chamber. Normal saline solution is boiled as above and used at the same temperature as the body. The apparatus is very simple: an ordinary Bohemian flask with a perforated indiarubber cork, a central perforation for the thermometer, and two others supporting

two glass tubes—one bent at an obtuse angle and projecting only a short distance below the cork, the other bent at an acute angle extending down into the sterilised water and connected outside with a broad flat nozzle, by means of india-rubber tubing. The water is made to run out through the nozzle by blowing down the short tube; the bottle can be raised or lowered according to the pressure required. It is of great importance to have the nozzle thoroughly aseptic; it should be made of platinum, and must be heated in a spirit-flame each time before it is used. The soft sticky lens matter is easily washed out, leaving behind the desired black pupil. I have not seen any harm attend irrigation, and its value cannot be doubted.

Accidents and Immediate Complications.—1. *Wrong position of the knife.*—The operator may find that he has introduced the blade of the knife with its cutting edge downwards instead of upwards. In case of this awkward occurrence, the knife must be cautiously withdrawn on the flat, so as to avoid much escape of aqueous. If only a little aqueous is lost, the knife may be again introduced, either at the same place, or by making a fresh puncture; if much aqueous has escaped, so that the iris is bulging forwards against the cornea, the operation had better be postponed for a day or two, in order to allow time for re-secretion of the aqueous; without this, the reintroduction of the knife and the upward section would cause an irregular wound of the iris.

2. *Early escape of the aqueous.*—Having completed the puncture and the counter-puncture, the section must not be made too slowly, or the aqueous escapes, and the iris bulges forwards in contact with the edge of the knife before the section is finished. Such an accident is not very serious, as the iris is often excised in the second stage of the operation; it is, nevertheless, much better to avoid its occurrence, because the outline of the excised portion of iris is likely to be jagged and less regular than when the iridectomy is made with scissors, and the hæmorrhage is likely to be troublesome in the succeeding steps of the operation.

As the counter-puncture is being made, there is sometimes a rush of aqueous into the subconjunctival tissue, which causes

the conjunctiva in its vicinity to start forwards in the form of a bladder, which obscures the point of the knife. This should be disregarded, and the blade of the knife pushed on in the horizontal direction until its point has passed through the conjunctiva.

3. *Hæmorrhage into the anterior chamber.*—The iridectomy is liable to be followed by hæmorrhage into the anterior chamber. The extravasated blood in this case comes partly from the iris and partly from the vessels in the neighbourhood of the canal of Schlemm; it usually ceases to flow after a few seconds, and should, if possible, be evacuated from the anterior chamber before the operation is proceeded with. This can usually be effected by gentle pressure with the end of the curette upon the posterior flap of the wound, or by gently stroking the cornea from below upwards with the back of the same instrument. If the bleeding cannot be stopped by these means the operation must be proceeded with. Although the capsule is now rendered invisible by the existing blood in the anterior chamber, it must still be lacerated with the cystitome, and the lens removed in the ordinary way. It usually happens that the blood escapes, and the hæmorrhage ceases with the removal of the lens. Adrenalin (F. 8A) prevents this liability.

4. *Difficulty in removing the cataract.*

(a) *Dislocation of the lens.*—If too great pressure is made on the lens in lacerating the capsule, and occasionally without any fault of the operator, the suspensory ligament is ruptured; the lens may then immediately sink back into the vitreous, or this may not occur until pressure is made with the view of causing it to present; vitreous at the same time often appears in the wound. This backward dislocation of the lens is one of the most serious accidents that can occur during a cataract operation; not a moment should be lost in passing the large scoop (fig. 93) into the eye well behind the presumed position of the lens, and attempting to extract it in its capsule. A good deal of vitreous is generally lost, but if the lens is extracted a very fair result may be obtained.

(b) *The wound may be too small.*—When this is the case the edge of the cataract may be seen to present between the lips of the wound, whilst the remainder refuses to come through.

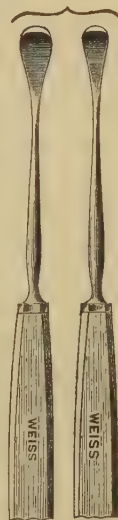
Under such circumstances, the section had better be enlarged at one or both extremities with small blunt-ended scissors; by making extreme pressure on the globe, the contusion of the iris and cornea in the region of the wound is liable to be followed by inflammatory trouble; while, by endeavouring to squeeze the lens through too small an opening, its cortical portion is likely to be scraped off and to remain in the eye. Sometimes when the lens appears in the wound during the pressure with the curette, its exit may be facilitated by gentle leverage. The assistant may be able to make traction upon it by means of the cystitome, or a small hook.

(c) *The capsule may be incompletely lacerated.*—Here the lens does not present at all. The use of the cystitome must be repeated.

5. *Escape of vitreous.*—This is always a serious complication, but the consequences of its occurrence will depend in a great measure on whether it occurs before or after the extraction of the lens. The presence of vitreous in the wound is indicated by the appearance of a perfectly transparent viscid fluid.

(a) If it occurs *before* the extraction of the lens, it is generally due either to the counter-puncture having been made too far from the cornea, or to too great pressure having been employed, either with the cystitome or with the curette in the fourth stage. If the vitreous is abnormally fluid, this acts as a predisposing cause. However the escape is caused, all pressure must be at once abandoned, the speculum removed, and a lid retractor (fig. 26) substituted for it; the lens should then be immediately removed with the scoop; if it still lies in its capsule, the latter must be removed with it.

The scoop (fig. 93) is introduced through the wound, and, with slight lateral movements, directed downwards and slightly backwards, so as to insinuate it behind the lens; when it has reached the lower edge of the latter, its handle is slightly depressed, and it is then gradually withdrawn, with the hope



FULL SIZE
FIG. 93.
Critchett's
Cataract
Scoop.

of bringing out the cataract at the same time. In case of failure in this method of traction, further attempts must be made so long as the cataract can be seen through the cornea.

(b) If vitreous *follows* the escape of the lens, it is due either to the latter having been expressed too suddenly, to a weak posterior capsule, or to compression of the globe by contraction of the ocular muscles. Very slight spasm of these muscles is liable to cause evacuation of the greater part of the vitreous humour. In order to prevent this accident, the eye should be kept thoroughly under the influence of cocaine, for, besides its anæsthetic properties, cocaine widens the palpebral aperture and temporarily arrests involuntary nictitation (blinking). The speculum should be either held forwards by the assistant or removed altogether, and the lids then separated by the surgeon's fingers. If vomiting occur at this period, the eyelids must be closed, and supported by a compress of cotton-wool during its continuance. The treatment to be followed will depend on the amount of vitreous which escapes. If this is considerable, not a moment should be lost in removing the speculum, closing the eyelids, and applying the pad. If only a small bead presents in the wound, separating its lips, the projecting part may be cut away with scissors; many operators, however, prefer to close the eye at once and trust to the vitreous falling back.

The loss of a small quantity of vitreous is not a serious accident; in fact, a considerable portion may escape without any *immediate* ill effects; but this is often followed at a later date by detachment of the retina, and consequent loss of vision.

The After-treatment and Remote Complications of Cataract Extraction.—Immediately after the operation the margins of the lids should be gently freed from any blood-clot, and a light dressing applied. This may be either of sterilised plain gamgee tissue or of double cyanide tissue. A piece $2\frac{1}{2}$ inches by $1\frac{1}{2}$ inch is sufficiently large, and should have its edges rounded; it is kept in its place by two or three pieces of adhesive plaster, but great care must be taken to exert no pressure upon the eyeball. The other eye should be closed in a similar manner. The patient must be carried back to his bed, which should be placed in a darkened room, the head of the bed being towards the window. It is necessary that visitors

and anything likely to cause excitement should not be allowed for twenty-four hours. A wise precaution is to tie the hands down to the sides of the bed, so that the eyes be not accidentally rubbed as the patient is waking from a sleep. No food or drink should be given during the three hours following the operation; should thirst be complained of, the patient may be allowed to suck a small lump of ice. After that time a light diet of beef-tea, fish, and farinaceous food may be given. After the first day ordinary nourishing diet may be ordered. Alcoholic drinks are not necessary, but a small allowance may be made if the patient cannot sleep without it. Should diabetes be present, a diabetic diet must be substituted.

The longer the dressing is untouched the better, and it is rarely necessary to change it before the third or fourth day after the operation, if an iridectomy has been performed. If, however, pain be complained of, the dressing should be carefully removed before this, and the lower lid slightly depressed with the fingers, in order to give vent to any pent-up tears. Pain with swelling of the lid necessitates examination of the eye itself. If iridectomy has not been performed, it is absolutely necessary to examine the eye on the morning following the operation, inasmuch as prolapse of the iris may have occurred, necessitating immediate iridectomy. If this operation be deferred, irido-cyclitis will almost certainly ensue.

In the normal course of events, on the third or fourth day the patient may be dressed and allowed to rest for a few hours on a couch, or on the outside of his bed. Atropine should now be instilled into the eye once daily as a precaution against iritis. On the fourth or fifth day the pad may be removed from the sound eye, a large double shade over both eyes being worn instead. The patient should be kept indoors for a fortnight; afterwards he may go out, wearing protective goggles of dark neutral tint, a pad of gamgee being placed in front of the aphakic eye. The atropine and the pad should be worn as long as any redness is present. About the sixtieth day the eyes will have reached the maximum of visual acuteness; they may then be tested for correcting glasses. The removal of the crystalline lens has rendered the eye exceedingly hypermetropic, and has

destroyed the power of accommodation. The patient will therefore require two pairs of convex spectacles for the purposes of distinct vision—the one to render the eye emmetropic, which will enable him to see all distant objects clearly, and the other to render him myopic, so that he may be able to read small print, or to do fine work at 20 to 40 cm. from the eyes. The strength of the lenses required for these purposes is usually about 10 D and 14 D respectively; but this will of course vary with the refraction of the eye (see Refraction). Astigmatism is usually present after cataract extraction; there is some bulging in the neighbourhood of the wound, which causes a certain amount of flattening of the cornea in the vertical meridian. Javal's ophthalmometer (see p. 544) is of great use in determining the amount of astigmatism present, which is necessarily all corneal.

The use of the spectacles should be gradually acquired, commencing with about half an hour's practice daily.

The slight pain arising from the operation usually ceases in the course of a few hours; its disappearance is always a favourable sign. On removing the dressings during the first few days succeeding the operation, the absence of pain in and around the eye, of any swelling of the lids, and of any muco-pus, is always a guarantee that the eye is progressing favourably. If the pain should reappear towards night, and become continuous so as to render the patient restless and uncomfortable, some sedative should be given, in order to procure sleep.

The *occurrence of severe and increasing pain* during the first few days after the operation is always an indication of some complication in the process of healing, and is sufficient to justify an immediate examination of the eye. The lids should be carefully separated, and the wound and other parts examined by means of focal illumination from the light of a single candle. We may thus find that the pain is simply due to accumulated tears, to an inverted lower lid, to the presence of eyelashes within the palpebral fissure, or to the commencement of inflammation.

Iritis is a frequent complication of cataract extraction; if an iridectomy has formed part of the operation its effects are less injurious than in the old flap operation, where it was the

cause of a large percentage of failures. The most usual time for it to come on is about the fifth day after the operation. Its presence is indicated by photophobia, œdema of the lids, pain, and chemosis; there is also copious lachrymation, but not muco-purulent discharge; the cornea may be clear, but the aqueous is turbid, and the iris somewhat changed in colour. In such a case a few leeches should be applied to the temple, 1 per cent. solution of atropine dropped into the eye three or four times daily, and the eye kept constantly warm by a large pad of cotton-wool over the closed eyelids. The extent of the damage the iritis may bring about will chiefly depend upon the amount of plastic exudation thrown out into the pupillary area; the amount of this exudation may be so great as to cause occlusion of the old pupil and of the new one formed by the iridectomy; the thick membrane thus established may also contract and draw the iris upwards towards the cicatrix, so as to diminish and displace the pupil. The inflammation may also extend to the rest of the uveal tract, setting up cyclitis or choroiditis, which may lead to complete loss of vision.

Entanglement of the iris in the angles of the wound is not uncommon where iridectomy has been performed. It is indicated by the presence of black nodules in the wound; these are of variable magnitude, and may be so extensive as to impede union, and even to form small cysts within the cicatrix. The entangled iris may also cause serious trouble by dragging upon the wound during contraction; this, again, may retard the healing process, and is often the cause of *recurrent iritis*. It may further be the means of setting up plastic *irido-cyclitis* in the operated eye; and this, as we have seen (p. 213), may extend to the second eye, and so set up *sympathetic ophthalmitis*.

The means of preventing this entanglement of iris at the time of operation have already been pointed out (p. 398); sometimes, however, this condition supervenes on the second or third day. If a knuckle of iris does appear, and especially if it evinces a tendency to increase in size, and to cause irritation of the eye, an attempt must be made to remove the prolapsed portion. This must be seized with forceps, and cut off

level with the globe by means of iris scissors. The eye must be kept closed with a light compress for at least a week after this, in order to favour the consolidation of the cicatrix. Should there be a recurrence of the prolapse after the operation, it can be lightly touched from time to time with nitrate of silver.

Persistent lachrymation and photophobia may be very troublesome, and may exist without any iritis. These usually disappear if the patient be kept indoors with the eye covered. Occasionally some remaining opaque lens matter appears to have an irritating action, and these symptoms are relieved by a needling operation to aid absorption.

Suppuration is attended by violent and increasing pain in and around the eye, by swelling of the eyelids, chemosis, and a copious muco-purulent discharge. It may commence at any time during the first few days following the extraction. When the lids are separated, and the eye examined during the early stage, the ocular conjunctiva is found to be distended with serum, the cornea is hazy, the edges of the wound present a greyish-yellow appearance, indicating the formation of pus, and hypopyon may be present. Unless this process can be immediately checked, it will extend to the whole of the cornea, to the tunica vasculosa, and to the vitreous, thus constituting severe panophthalmitis, which must terminate in the destruction of the globe. No time, therefore, must be lost in endeavouring to reduce the inflammation. The eyelids should be widely separated and the discharge well washed away with an antiseptic lotion every hour during the day, and every two hours during the night; after each ablution the outsides of the eyelids and surrounding parts should be well fomented with boracic acid or perchloride of mercury. Good nourishing diet, port wine or brandy, quinine or bark and ammonia, should be given internally, with opiates if necessary. By these means the affection may take on a less destructive form, and may occasionally be arrested before total destruction of the eye has taken place.

Intra-ocular hæmorrhage from the ciliary, choroidal, or retinal vessels may come on immediately or shortly after the operation. The extent of the hæmorrhage varies very con-

siderably. Its advent may be marked by severe pain; the globe is seen to be filled with blood, which escapes through the wound and oozes through the dressings. Such an eye is sure to be lost, and may require immediate excision on account of the pain and the bleeding. On the other hand, the hæmorrhage may be only from the ciliary vessels, being confined to the area occupied by the lens. If this is the case, complete absorption will probably take place in from one to three months. A small subretinal hæmorrhage may clear up, but on the other hand may cause total and permanent blindness.

Spasmodic entropion is a troublesome complication which is apt to come on a few days after the operation. The lax state of the tissues acts as a predisposing condition, while the operation-wound, and possibly the compressing bandage, excite contraction of the orbicularis muscle. Unless this condition is quickly remedied, the irritation set up by the inverted lashes of the lower lid brushing against the cornea is very likely to lead to loss of the eye.

Treatment.—Sometimes it is sufficient to substitute a large shade for the bandage; if this is ineffectual or undesirable, the lid should be drawn down, and the face just below the eye well covered with a film of contractile collodion. Adhesive plaster is frequently more effective than collodion. If this fails—and it seldom does if properly applied—a fold of skin must be at once excised, as described on p. 32.

Delay in the healing of the corneal wound may occur even though all symptoms of inflammation and irritation are absent. The wound may gape, and if so the size of the dressing should be increased and slight pressure exerted. A conjunctival flap (see p. 394) is a safeguard against this complication.

Cystoid degeneration of the cicatrix may occur after the peripheral operation with iridectomy. The iris is, as a rule, more or less entangled in the wound. It is usually due to a glaucomatous condition of the eye.

Secondary glaucoma may follow cataract extraction a considerable time after the operation. This may be brought about in one of several ways. A serous cyclitis may develop and cause a block at the iridic angle, owing to exudation

between the fibres of the ligamentum pectinatum. The glaucoma, however, may occur where little or no inflammation has developed, even though a preliminary iridectomy has been performed. This is probably due to an adhesion between the corneal scar and the lenticular capsule, or the root of the iris. Glaucoma is, however, more common after simple extraction.

Erythropsia is a frequent and annoying remote complication of cataract extraction. It is usually temporary, and soon passes off after the refractive error has been corrected. Its cause is obscure. It may be that a vaso-motor dilatation of the blood-vessels is in some way produced, either by the diminished intra-ocular pressure or by the diffuse light stimulus. It may occur in eyes where the sight with correcting glasses is normal.

Opaque Capsule. Secondary Pupillary Membrane.

If the anterior layer of the capsule of the lens has been properly lacerated, a large triangular gap generally remains; sometimes, however, owing either to the laceration having been insufficient, or to the capsule floating back over the pupil, a layer is left which interferes with vision; sometimes the capsule is so transparent that it can be seen only by very careful focal illumination or by the use of the ophthalmoscope with a +12 D lens; but even in these cases it causes considerable interference with vision, probably because it is always slightly wrinkled. In other cases the capsule forms an opaque membrane, which can be distinctly seen with the naked eye. It is extremely doubtful whether the capsule itself ever becomes opaque. It is certain, however, that most cases of so-called opaque capsule are really composed of opaque cortex adhering to the anterior portion of the capsule, which is itself transparent. Hence the prevention of what is sometimes termed secondary cataract depends chiefly on the thoroughness with which the cortical portions of the cataract were removed at the time of the extraction. Occasionally, a pupil which was quite clear at the time of the operation and some weeks later, subsequently presents a secondary cataract; this is due to the cataract being immature at the time of the

original operation, with retention of some portion of the cortex.

The membranes which form in the pupil as a consequence of iritis are of much more serious importance. They are generally thick and tough ; they adhere by their margins to the iris, and by their contraction tend to narrow the area of the pupil.

Treatment.—No operative measures must be had recourse to until all signs of active inflammation have subsided, and until no further absorptive action is likely to take place. Operation for secondary cataract is rarely necessary until from two to four months after the extraction.

Thin membranous forms can be readily torn through with a single cataract needle. The pupil should be first dilated widely by means of atropine. The needle is introduced about 2 mm. from the sclero-corneal junction. After the operation the iris must be kept at rest for a few weeks with atropine, a weak solution being all that is necessary. For tougher opacities two needles should be used, as suggested by Bowman, and the opening made by tearing from the centre.

For the still denser membranes formed by lymph, or lymph and capsule, needling is often not sufficient ; it is difficult in such a case, even with two needles, to avoid making some traction, and if inflammatory symptoms follow, the opening made generally gets closed by fresh lymph. By far the most effectual proceeding is to divide the membrane, and the iris if necessary, with scissors, of which a good pattern is that devised by Carter. This operation is called *iridotomy*, and is described on p. 236.

DISLOCATION.

Dislocation of the crystalline lens may be congenital, spontaneous, or traumatic.

Etiology.—When *congenital*, it is often hereditary, and is due to deficient formation of the suspensory ligament, which deficiency is in its turn usually the result of imperfect closure of the choroidal fissure. It is therefore, as a rule, the lower

and inner part of the zonule which is maldeveloped, and consequently the lens is drawn upwards and outwards by the traction of the ligament in this situation. The luxation is, as a rule, partial, and generally occurs in both eyes; it is often combined with a coloboma lentis, or some other form of maldevelopment. When *spontaneous*, it is usually the result of pathological degeneration of the vitreous humour and of the suspensory ligament. It is more commonly found amongst diseases in which these structures are known to be affected, as in sparkling synchysis, high degrees of myopia, staphyloma of the ciliary region, &c. When *traumatic*, it is usually the result of a contusion of the globe, which has caused rupture of the suspensory ligament.

Symptoms.—The symptoms vary with the extent of the displacement.

In *partial dislocation*, by using the ophthalmoscope mirror the edge of the lens can be seen as a narrow dark line, slightly curved, crossing the peripheral part of the pupil. The appearance presented by the lens margin is quite unmistakable, and is diagnostic of dislocation of the lens, as, even in extreme dilatation of the pupil, it can never be seen when the lens is *in situ* (*vide* figs. 1 and 2, opposite p. 366). With focal illumination (p. 110) the lens can often be distinguished by a sort of greyish opalescence, the edge appearing as a golden line. When the displacement is such that the edge of the lens extends to the visual field, the symptoms are more numerous and pronounced. The surface of the iris is seen to be irregular, one part being more or less bulged forwards towards the cornea, whilst the remainder is depressed; this depressed portion may also be tremulous when the eye is moved. A tremulous iris (*iridodonesis*) is always a sign of a dislocated lens, whether the displacement be partial or complete. Absence of the lens is not always attended with iridodonesis, as may be noted in some cases of cataract extraction. The patient often complains of monocular diplopia. The visual acuteness is also greatly impaired, the oblique position of the lens having produced irregular astigmatism, which cannot be corrected by spectacles. There is often high myopia in cases of congenital subluxation, which may

be entirely lenticular, and must therefore be due to increase in the convexity of the crystalline lens resulting from a slackening of the capsule, or to an arrest in the normal flattening of the lens during its growth. The power of accommodation is very defective. When the pupil is fully dilated with atropine it is often found that by using a stenopaïc disc the double vision of the eye is dispersed, and that the vision is different when the slit is held in front of the partly dislocated lens from that which is obtained when it is held in front of the part where the lens is absent; in the latter position the eye is found to be highly hypermetropic. On examining the fundus with the ophthalmoscope, either by the direct or indirect method, two images of the optic disc and retinal vessels are seen; this phenomenon, as well as that of the monocular diplopia, is explained by the fact that the rays passing through the lens and those passing outside it have different foci.

In *complete dislocation* the lens falls either backwards into the vitreous or forwards into the anterior chamber. In *dislocation into the vitreous*, this substance, being more liquid than normal, allows the lens to sink to the bottom of the chamber. In this new position it gradually becomes opaque; by focal illumination it may sometimes be seen, and with the ophthalmoscope it appears as a dark floating mass at the bottom of the cavity when the eye is moved. The iris, having lost the support of the lens, falls somewhat backwards, and undergoes a tremulous motion when the eye is moved. The refractive condition of the eye is the same here as it is after cataract extraction.

In *dislocation into the anterior chamber* the lens in its capsule passes forwards through the pupil and becomes wedged between the iris and the back of the cornea. The appearance presented by the transparent lens in the anterior chamber is that of a drop of oil. The iris is pushed backwards, the pupil somewhat dilated; the refraction is myopic, due partly to the forward displacement of the lens, and partly to an increase in its convexity owing to the restraining influence of the capsule being absent. The power of accommodation is abolished.

Dislocation into the anterior chamber may be primary or

secondary. A blow on the peripheral part of the cornea may cause a tilting backwards of one side of the lens, and a corresponding tilting forwards of the opposite side, which may slip over the edge of the iris. On the other hand, a partial dislocation upwards or downwards may, secondarily, during an act of stooping or straining, become a complete luxation forwards. Sometimes a congenital dislocation upwards, under atropine, may be transformed into an almost complete luxation forwards, with a corresponding increase in the amount of myopia. The lens may remain for some time in the anterior chamber without becoming opaque, and without causing pain. Subsequently, however, the iris becomes paralysed and dilated, glaucomatous symptoms arise, the lens becomes cataractous, and more or less severe plastic inflammation of the iris supervenes.

When dislocation arises from an injury it is frequently accompanied by other lesions, such as rupture of the choroid and of the sclerotic; hæmorrhage may also take place either into the fundus, or into the anterior chamber, or both. Sometimes the lens escapes from the globe altogether through a wound in the sclerotic, and may be discovered beneath the ocular conjunctiva; the commonest place where the lens leaves the globe is the upper sclero-corneal junction.

Treatment.—1. *When the luxation is partial* the treatment which should be adopted will depend on the amount of displacement and the interference with vision. When vision is not much impaired, and the lens always remains in the same position, no treatment is advisable. These partial luxations, however, often become complete, the lens falling forwards into the anterior chamber or backwards into the vitreous.

When the displaced lens is transparent and its position permanent, but vision is seriously interfered with, some improvement may sometimes be obtained by making an artificial pupil in the direction towards which the lens is displaced; the results, however, are uncertain, and apt to be disappointing.

When the lens is opaque, and in a young subject, an attempt may be made to get rid of it by needling; but in a person over thirty-five it had better be extracted by the method of von Graefe (p. 390).

2. *When the dislocation is complete* the lens is useless, and its presence is liable to cause an attack of glaucoma ; hence its removal should be undertaken when this can be done without much risk. The removal of the lens is especially indicated in cases in which inflammatory symptoms have already appeared. Unfortunately, the removal of the lens from the vitreous involves the loss of so much of that fluid, while the difficulty in extracting the lens is so great, that the operation is attended with considerable difficulty ; an attempt, however, should be made to remove it by means of a scoop or a vectis after the ordinary incision and iridectomy. If the lens cannot be delivered, or if the vitreous is all lost during the operation, it is better to eviscerate the globe (p. 146) at once, and so save the patient prolonged suffering and risk of sympathetic trouble.

When the lens lies in the anterior chamber it may be removed either by needling and solution or by linear extraction ; the former methods are only adapted for children, and, on account of secondary glaucoma usually set up, must be followed by paracentesis or suction. It may be necessary to remove the capsule later ; this can be done by seizing it with fine forceps, introduced through a small wound.

In young adults, where the lens is fairly soft, the corneal incision may be made with a keratome, whose point is made to pass through the capsule of the lens. The nucleus may now be removed with a scoop or small vectis, and the cortex carefully extracted with a curette or left to become absorbed.

In older persons, the lens, being comparatively hard, is very liable to slip back into the vitreous while the incision is being made, the hyaloid membrane being ruptured ; hence it is generally desirable to have the pupil contracted by eserine, and to fix the lens, by a needle passed through the cornea, while making the incision.

CHAPTER XII.

THE VITREOUS HUMOUR.

ANATOMY—MUSCÆ VOLITANTES—VITREOUS OPACITIES—SYNCHYSIS—CYSTICERCUS CELLULOSÆ—HÆMORRHAGES—CONGENITAL DEFECTS.

ANATOMY.

THE vitreous body or humour is the transparent jellylike substance which occupies the whole of that part of the globe which lies behind the lens and its suspensory ligament. The crystalline lens rests in a depression on its anterior surface (the *fossa patellaris*), and the attachment of the vitreous to the posterior capsule is firmer than elsewhere. Traversing the vitreous, from the optic nerve to the middle of the posterior capsule, is a canal of about 2 mm. diameter—the hyaloid canal (*Stilling's* or *Cloquet's canal*). The consistence of the vitreous gradually becomes less firm as age advances; in adult life it is a viscid fluid, somewhat more tenacious than the uncoagulated white of egg. Its index of refraction is 1.336, and therefore almost identical with that of the aqueous humour.

When hardened in chromic acid, or by freezing, the vitreous shows a tendency to split into concentric layers in its peripheral portions, while the central part shows a less marked radial striation. There is some doubt as to whether the lamellæ thus formed correspond with any structural arrangement of the solid constituents, although it was formerly thought that such was the case. Recently it has been shown that the vitreous humour consists of a semifluid portion contained in a framework of concentrically arranged septa. The more fluid portion contains a few characteristic vitreous cells; these are of a roundish shape, somewhat larger than white blood-corpuscles, and contain one, two, or three perfectly transparent vesicles which nearly fill up the cavity. In the peripheral portions of the vitreous, stellate and fusiform cells are also found, which contain similar round transparent vesicles. The outline of the cells can be made more apparent if a portion of recent vitreous is stained

in a weak solution of logwood. If examined on the warm stage these vitreous cells are found to exhibit amœbiform movements.

In the fœtus the vitreous contains many small branching fibrils with small granules upon them resembling nucleoli rather than nuclei. Some of these fibrils may persist throughout life. The vitreous fluid is completely enclosed by a fine homogeneous structure, the *hyaloid membrane*, which separates it from the retina, the papilla, the ciliary body, and the lens capsule. The nutrition is carried on chiefly by the ciliary processes, slightly by the retinal vessels.

Muscæ volitantes.—Under ordinary conditions the cells which float in the vitreous do not give rise to any visual sensation, although shadows must be thrown by them upon the retina. This is probably because, in the first place, the mind is accustomed to disregard them; and, secondly, the shadows are much less defined than the images of external objects. If, however, the light enters the eye in an unaccustomed manner, as when a strongly diverging pencil of rays is employed, as is the case in looking through a pinhole aperture held close to the eye, they become visible, especially if the eye be directed to a large white surface, such as a white cloud, so that there are no other retinal images with which to compare them. Occasionally, owing either to hyperæsthesia of the retina, or to an error of refraction which impairs the definition of the retinal images of all objects, the shadows of the vitreous cells become visible by ordinary light, and then constitute the troublesome symptom known as *muscæ volitantes*. In this condition the vision is unimpaired, but the patient is often much alarmed by the *muscæ*, which he looks upon as an indication of impending blindness. In reality they are of no importance whatever, except in so far as they indicate the necessity of examining for any errors of refraction, and improving the general condition. Another cause of *muscæ volitantes* may be the persistence of some of the fibrils found in fœtal eyes.

VITREOUS OPACITIES.

Opacities in the vitreous may be floating or fixed. The free opacities are usually multiple and of small size, while the fixed, which are less common, are more often single, and assume the form of a membrane. Both forms are usually due to the exudation of inflammatory material, and are generally secondary to

disease of the ciliary body or choroid. Cases, however, are frequently seen in which no cause whatever can be found for the opacities, the site of the inflammation being out of the range of the ophthalmoscope, or the inflammatory changes being too fine to be visible.

In order to ascertain if there are any opacities in the vitreous the plane mirror should be employed; if this be held at a distance of 8 or 10 inches from the eye, and the patient move the latter successively in different directions, any opacities in the vitreous, unless they are extremely minute, will come into view; if not seen by this method the mirror should be held quite close to the eye, and convex lenses of gradually increasing strength be placed behind it, so that different parts of the vitreous are successively brought into view, from the deeper to the more superficial layers. If the examination be conducted in this manner, the presence of vitreous opacities can hardly be overlooked.

To determine the situation of an opacity in one of the media of the eye, the direction of the movement of the opacity in relation to the movement of the eyeball must be carefully observed. If it is behind the centre of curvature of the cornea (*i.e.* a point 0.63 mm. behind the posterior surface of the lens), it will appear to move in a direction opposite to that of the globe; the contrary is the case if it is in the anterior part of the vitreous, in the lens, or in the cornea.

Floating opacities.—These are usually of very small size, but occasionally there are mixed with the smaller ones a few of larger size, which are probably formed by their coalescence; the latter always appear black when viewed with the ophthalmoscope, because they intercept the light reflected from the fundus; but if, as occasionally happens, an opacity is sufficiently far forwards to be seen by focal illumination, it appears white or greyish.

The *fixed membranous opacities* usually present sufficient surface to reflect light thrown into the eye, and so appear white; they are much rarer than the small floating opacities, of which they are probably in many instances a further development. Occasionally vessels can be seen running on them for a short distance.

Opacities of the vitreous are met with in the following conditions. *In myopia of high degree complicated with posterior sclero-choroiditis* we frequently find flocculi floating about in the unnaturally fluid vitreous. They are usually few in number; as a rule they do not interfere greatly with vision, and need not give rise to much anxiety; but should they be numerous and the vision much impaired, a guarded prognosis must be given, as this condition may be the forerunner of detachment of the retina. *In choroiditis* where the pigmentary layer of the retina is thick, the appearance of numerous floating opacities in the vitreous is sometimes the only symptom of the inflammation. In severe choroiditis and cyclitis membranous opacities occasionally form, which completely prevent any reflex being obtained from the fundus. Syphilitic retino-choroiditis has already been mentioned (p. 203) as being accompanied by opacities in the vitreous. *As a result of hæmorrhage into the vitreous*, membranous opacities usually persist, if the hæmorrhage was at all extensive. In these cases the opacity may be fixed, and may take on the form known as retinitis proliferans (see p. 289). *After cataract extraction*, when the patient presents himself for glasses, it is very frequently found that the vitreous contains floating membranous opacities, which diminish the efficacy of the operation. These are due to degenerative processes taking place in the vitreous concurrently with the development of the cataract.

DEGENERATIVE CHANGES.

Synchysis.—**Abnormal Fluidity.**—Occasionally, in old persons, especially if they are myopic and a posterior staphyloma is present, the vitreous becomes unnaturally fluid without any other morbid change being apparent in it; unless there are floating opacities also, this condition cannot be diagnosed, but its presence may complicate the operation of cataract extraction by predisposing to an escape of vitreous. Choroiditis is also a cause of synchysis.

Synchysis scintillans is the term applied to a variety of softening of the vitreous, in which a number of brilliant floating particles are observed. When the ophthalmoscope is used they

look like floating spheres or discs of gold moving in all directions. They are extremely numerous in the anterior layers of the vitreous. When the pupil is dilated they can be seen sometimes by the oblique focal illumination, as well as by the ophthalmoscope. On careful examination two kinds of particles may be seen : the one, small and white, composed of *tyrosin* ; the other, larger and more lustrous, consisting of *cholesterin*. Crystals of phosphates are also sometimes present. The vitreous may be rendered so opaque by the existence of these bodies that no detail of the fundus beyond can be obtained. The affection is mostly observed in old people. It may exist for some time without causing great visual trouble.

CYSTICERCUS CELLULOSÆ.

Cysticercus cellulosæ, the scolex of the *Tænia solium*, is occasionally found in the vitreous in Germany, but in this country it is almost unknown. It is generally developed beneath the retina, and, after having perforated that membrane, projects into the vitreous. When the media are clear, the parasite can be seen with the ophthalmoscope as a bluish-white semi-transparent cyst ; it moves about with the slightest deviation of the eye, and possesses certain undulating movements of its own, upon which depends its diagnosis. Hill Griffith gives the following description of the cases he had seen : ‘ In each case one saw with the ophthalmoscope a very large spherical bluish-white cyst, and springing from this the neck of the animal, like an alabaster pillar, surmounted by the head and suckers, which, with its slow, regular, and graceful movements, reminded one of an elephant’s trunk, the whole appearance presenting a picture never to be forgotten.’ Its presence is usually followed by loss of the eye, which becomes disorganised and atrophied.

HÆMORRHAGES.

Hæmorrhages into the vitreous are usually caused by injury, as a direct blow or wound of the eye, or by concussion propagated through the skull. Sometimes, however, they are not traumatic in origin, when their etiology resembles that of

retinal hæmorrhages. Vicarious menstruation has been known to take place regularly into the vitreous. Recurrent vitreous hæmorrhage may occur in young adults (see p. 281). Occasionally no cause can be determined.

Symptoms.—The hæmorrhages announce themselves by partial or total darkening of the field of vision; this may come on gradually, or occur suddenly. The extravasations can usually be seen with the ophthalmoscope, and frequently also by the oblique focal illumination. Sometimes, however the extravasated blood cannot be seen, neither can any red reflex be obtained; the pupil looks black both upon ophthalmoscopic examination and by oblique focal illumination. The presence of Sanson's images (see p. 374) will show that the opacity is behind the lens; besides, a cataract is rarely so black as to be mistaken for this condition. Erythropsia is occasionally present.

The hæmorrhages may disappear in the course of a few weeks, but more frequently are followed by pigmented floating opacities. They may lead to retinitis proliferans, or to retinal detachment.

Treatment.—Both eyes must be rested, no reading or writing being allowed, and neutral-tinted glasses being worn. Mercurial inunctions should be used on the forehead, and iodide of potassium taken internally. The diet and habits of the patient must be carefully regulated, and any constitutional disease treated. The bowels must be kept loose by mild aperients.

CONGENITAL DEFECTS.

Persistent Hyaloid Artery.—See p. 255.

Congenital Membranous Opacities.—An abnormal development of the mesoblastic tissue which forms the vitreous fluid may produce coarse fibrous membranous striæ throughout the vitreous chamber. The importance of the condition lies in the great difficulty in diagnosing it from glioma retinae (see p. 308).

CHAPTER XIII.

GLAUCOMA.

PRIMARY GLAUCOMA—SECONDARY GLAUCOMA—CONGENITAL GLAUCOMA,
OR BUPHTHALMOS.

GLAUCOMA is the name given to the group of symptoms caused by an excess of intra-ocular tension. When it occurs independently of any other affection of the eye, it is called *primary*; when it is caused by pre-existing eye-disease, it is known as *secondary glaucoma*.

PRIMARY GLAUCOMA.

Etiology.—Primary glaucoma occurs somewhat more frequently in females than in males. It is essentially a disease of advanced life, 70 per cent. of the cases occurring in those who are over fifty. There is, however, a form of the disease, known as *buphthalmos* or hydrophthalmos, which is congenital. Cases of acquired glaucoma have been recorded in children. There are certain predisposing factors in the etiology of glaucoma.

1. It has been found by Priestley Smith that the normal cornea ceases to grow at the age of five years, whereas the crystalline lens is increasing in size throughout life. There is consequently some encroachment on the space in its immediate neighbourhood, with a corresponding diminution in the patency of the iridic angle.

2. The dimensions of the cornea bear a certain proportion to the dimensions of the whole eyeball. Small corneæ denote, as a rule, small eyeballs, and it has been found that the dimensions of the corneæ of glaucomatous eyes are below normal.

3. A large proportion of glaucomatous eyes (50 to 75 per

cent.) are found to be hypermetropic. It is very rare for a myopic person to be attacked with glaucoma.

The exciting causes of glaucoma are extremely obscure. There is sometimes a history of shock, or worry. Occasionally the disease is set up by some severe illness, such as influenza, small-pox, acute rheumatism. The local administration of atropine may start an attack; cases have been recorded where a single drop of homatropine has been the initial cause. Concussion with no dislocation of the lens, or rupture of the lens capsule, is said to have produced glaucoma.

The disease occurs in every degree of severity, and varies exceedingly in its rate of progress; it may be so acute as to terminate in total blindness in the course of twenty-four hours, or so chronic as to go on for months, and even years, before arriving at this condition. It is, however, always progressive, unless checked by remedial measures.

The **symptoms** may be divided into :

1. Those which are premonitory.
2. Those which accompany the actual attack.

Premonitory symptoms are seldom wanting, although they are frequently unheeded by the patient until the true onset of the attack. One of the earliest is the rapid impairment of accommodation—rapidly increasing presbyopia. The patient has been unable to read small print (No. 0.5 Snellen) without spectacles of greater strength than should be required at his age (see Refraction), and has found it necessary to increase the strength of the latter perhaps several times in the course of a few months. All artificial lights, such as the gas or candle flame, have at times been surrounded by a halo of brightness, or by coloured rainbowlike rings, the violet end of the spectrum being internal, the red end external. In some cases the patient complains of cloudiness of sight, which he describes as ‘fog,’ or ‘mist,’ before the eyes. This is not always present, but comes and goes at intervals; it is more likely to supervene after prolonged use of the eyes, and is therefore more common at night than in the morning. Occasionally the patient may find himself in total darkness for several seconds from sudden failure of vision. The paroxysmal nature of these premonitory symptoms is very characteristic.

Of the symptoms which accompany the actual attack, the most important are :

1. Increased intra-ocular tension.
2. Cupping of the optic disc.
3. Contraction of the visual field.
4. Dilatation of the pupil.
5. Pain and other symptoms.

1. *Intra-ocular tension is always increased* ; in fact, this symptom is pathognomonic of the disease. *In order to ascertain the degree of tension*, the patient should be directed to look towards the floor, whilst the head is retained erect ; the upper part of the globe is thus brought well forward, so that it can be reached by the tips of the surgeon's two index fingers, and so examined by gentle pressure through the upper lid. Considerable practice in this palpation is necessary before the *tactus eruditus* can be acquired ; the affected eye should be compared with the other, and with the normal eye of another person. The following method of indicating the amount of intra-ocular tension (Bowman) is now almost universally adopted :

T_n, normal tension.

T + ? tension probably increased.

T + 1 „ perceptibly increased.

T + 2 „ increased, but the globe can be dimpled.

T + 3 „ increased so much that the globe cannot be dimpled (stony hardness).

T - ? „ probably diminished.

T - 1 „ certainly diminished.

T - 2 „ much diminished.

T - 3 „ very much diminished (globe flaccid).

The increase of tension is almost in direct proportion to the severity of the disease : in the most acute cases it is usually very high (T = + 2 or + 3) ; in the subacute forms it is less increased (T = + 1 or + 2) ; and in the chronic varieties it may be only slightly augmented (T = + ? or + 1). This increased tension is not necessarily persistent, but, like the premonitory symptoms, is often intermittent. This is especially the case where the glaucoma is subacute or chronic, and often introduces a difficulty in diagnosis.

Plate XVI.

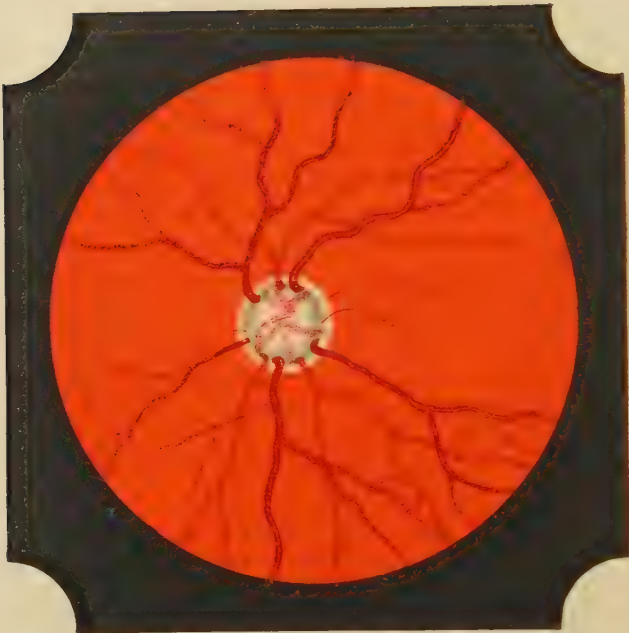


Fig. 1.—Deep Glaucoma Cupping (*edge of disc in focus*).



Fig. 2.—The same (*bottom of excavation in focus*).

W.J.P.

2. *Cupping of the optic disc* is not present in the earlier stage of glaucoma, but is always found where increased intra-ocular tension has existed for some time. It is directly due to this intra-ocular pressure, which causes a bulging backward of the lamina cribrosa, the entrance of the optic nerve being the weakest part of the walls of the eyeball. The depth of the cup is very variable; it is more marked in persons under fifty than in those above that age. Its floor presents a bluish-white appearance, the lamina cribrosa being plainly visible through the atrophied nerve-tissue; this is most pronounced in advanced cases.

The cupping can be best seen by the direct method of ophthalmoscopic examination; but atropine must on no account be used to dilate the pupil, as this invariably aggravates the symptoms. It is only, however, in the more chronic forms of glaucoma that the details of the fundus can be seen; in acute glaucoma the media are too hazy to admit of an ophthalmoscopic examination. Examined in this way, the cupped disc presents the appearance represented in figs. 1 and 2, on the opposite page. Fig. 1 is the condition seen when the vessels on the fundus are in focus. The vessels at the bottom of the cup are blurred and indistinct, for they are on a posterior plane and consequently out of focus. There appears to be a loss of continuity in the vessels at the margin of the disc; this is due to the vessels being hidden under the shelf of the excavation, around which they curve to reach the level of the fundus. In order to bring the disc or bottom of the cup into view, it will be necessary to interpose one of the concave lenses of the ophthalmoscope, the strength of the lens thus required to bring the lamina cribrosa into focus being proportionate to the depth of the cup (see fig. 2). In fact, we have only to allow 0·3 mm. for each dioptre of the lens used, in order to obtain an approximate estimate of this. Thus, suppose the edge of the disc and the vessels there to be in focus without any lens being interposed between the two eyes, and that a lens of 4 D is required to bring the lamina cribrosa into focus, then ($0\cdot3 \times 4 = 1\cdot2$) the approximate depth of the cup will be 1·2 mm. If the head be moved from side to side, the bottom of the cup being farther away

appears to move in the same direction as the observer's head; this parallax or change in the relative positions of the floor and edge of the cup is quite diagnostic.

The parallax can also be seen by the *indirect method*; in this case, if the lens which is used by the observer be moved through a small space in front of the eye, the images of the vessels at the edge of the cup, and those of the vessels at the bottom of the cup, appear to change their relative positions; those of the former seem to move more quickly than those of the latter. This phenomenon is easily explained. In fig. 94,



FIG. 94.—Optical Parallax.

let o be the position of the vessel at the edge of the disc, and o' that of a vessel at its bottom. Let i and i' be the respective images of these vessels. Then the distance Li is greater than Li' . If the lens be moved from L to L' , the image i' , being farther from the centre L' than the image i , will have to describe a greater space in the same time, and so i' will be displaced more quickly than i .

It is important to distinguish between the cupping of the optic disc which is due to increased intra-ocular pressure, and the physiological cupping already described on p. 257. By comparing fig. 1, opposite p. 254, with fig. 1, opposite p. 423, it will be seen that the physiological excavation occupies only a part of the area of the disc, whilst in glaucoma the whole

disc is depressed. The vessels are displaced towards the inner side of the cup.

Pulsation of the veins of the optic disc is always produced by increased tension, but, as it is very frequently present in healthy eyes, it is of little value as a symptom unless it can be proved that it did not previously exist. *Arterial pulsation* at the optic disc is occasionally seen, and when present is an important diagnostic sign. It is 'the expression of the contention between the pressures in the arteries and in the ocular chambers, and the alternate supremacy of each' (Priestley Smith). With the exceptions mentioned on pages 252 and 253, it is always due to increased intra-ocular tension or to aortic regurgitation. It may be present before the onset of an acute attack, and at any stage in the course of chronic glaucoma.

The optic disc is frequently surrounded by a ring of atrophied choroid, through which the sclerotic shows up as a white circle.

3. *Contraction of the field of vision* is always present in glaucoma. The field for white first commences to contract on the inner part, and then over the upper and lower portions of the periphery; from these inner, upper, and lower portions the obliteration gradually proceeds towards the point of fixation, which is ultimately destroyed, leaving only a contracted space in the outer part of the field in which vision still remains, although its acuity is necessarily much diminished.

It is an interesting fact, and one which is almost peculiar to glaucoma, that the limits of vision for colours follow the same kind of contraction as those for white (see Perimetry). Fig. 95 is a chart showing the visual field for white, blue, red, and green respectively, which was taken from a case of moderately advanced primary glaucoma. On comparing this with the normal visual field (fig. 62) it will be seen that all the areas for colours are contracted almost concentrically with that for white. So long as the central part of the field of vision—that is, the part which corresponds to the yellow-spot region of the retina—is not encroached upon, the patient may enjoy very good central vision both for white and for colours; he will, however, be unable to perceive objects other than those towards which his eye is directed; his vision is similar to that

of a person looking through a tube. After the obliteration has passed the central region the vision becomes very defective, and finally is lost altogether.

The above is the description of a typical glaucomatous field of vision; it is by no means rare to find less typical contraction.

4. *Dilatation of the pupil.*—In the *early stage* of primary glaucoma the pupil is always somewhat dilated; it is usually oval, and is moderately active. As the disease advances, the dilatation becomes greater, the activity is lost, the periphery of



FIG. 95.—Visual Field in Glaucoma (right eye).

— — — blue, red, green.

the iris adheres to the back of the cornea near the circumference of the latter. In very advanced cases the edge of the pupil forsakes its normal position against the capsule of the lens and becomes everted (ectropion iridis), so that a ring of brown pigment (uvea) is now seen to encircle the pupil in front. Finally, the iris becomes reduced to a narrow band of atrophied tissue.

This dilatation of the pupil is chiefly brought about by the lowering of the blood pressure, and partly by the paralysis and atrophy of the constrictor pupillæ.

5. Other important symptoms present themselves in glaucoma, but are less constant than those just mentioned.

Pain is sometimes a premonitory symptom. The actual onset of acute glaucoma is nearly always marked by intense pain in the eye and in surrounding parts, as the side of the nose, the temple, and the back of the head. The sudden appearance and extreme violence of the pain are important features in acute cases. Similar but less severe pain is sometimes present in subacute glaucoma, and occasionally in the chronic forms ; but the majority of the last are free from this symptom.

Inflammatory symptoms are always present in the acute and subacute forms, but are absent in chronic glaucoma. In the most acute cases there is intense congestion of the circumcorneal zone of vessels, and often of the whole conjunctiva ; there may be considerable chemosis of the ocular conjunctiva, and œdema of the eyelids. The iris loses its brilliancy, the aqueous and vitreous humours become turbid, and the cornea may be steamy. In subacute cases there is dusky redness of the vessels in the circumcorneal zone. The haze in the cornea is due to an œdema ; drops of liquid, very minute in size, collect in the anterior portion of the substantia propria, and immediately beneath the corneal epithelium. This œdema is the cause of the impaired vision, and of the haloes.

Shallowness of the anterior chamber is frequently found in cases of increased tension, but it is not a constant symptom ; the iris appears to be pushed forwards by advancement of the lens. In acute and subacute cases this forward bulging of the iris and lens is sometimes so pronounced that these structures appear to be in actual contact with the back of the cornea.

Impaired sensation of the cornea is a common symptom, and of great diagnostic value. When the tension is greatly increased, and especially when this has been of long duration, the cornea may be touched without exciting reflex contraction of the orbicularis, and without discomfort to the patient, owing to the compression and consequent paralysis of the nerve-filaments.

Opacities of the media are nearly always present in the acute and subacute, but are rare in the chronic forms of glaucoma. The cornea often becomes dull and 'steamy' in appearance, and small transparent vesicles may appear ; the

aqueous is turbid, and may contain small hæmorrhages: the vitreous frequently presents floating opacities. In old-standing cases the lens becomes opaque.

From what has been said of the symptoms of primary glaucoma it will be evident that an extensive range of cases is met with, and that, according to the nature of their prominent symptoms, they may be conveniently divided into three or four groups—viz. the *acute*, the *subacute*, the *chronic*, and the *hæmorrhagic*. The first three differ rather in degree than in kind, for intermediate forms occur, and a case belonging to one group may at any time assume the characters of another. The *hæmorrhagic*, however, presents marked differences in its cause, and in the effect of treatment.

In *acute cases* the actual attack is generally ushered in by severe pain in and around the eye, often extending over the whole side of the head; vomiting is not unfrequently present, and this, with the pain in the head, may cause the local trouble to be overlooked. The conjunctiva is usually intensely injected and covered by large tortuous veins. The pupil is inactive, semi-dilated, and oval. The cornea and media are always turbid; so that the iris looks muddy and the fundus cannot be seen. Tension is greatly increased, and vision becomes rapidly impaired, so that in the worst cases (*glaucoma fulminans*) total blindness may ensue in twenty-four hours or less.

The *subacute cases* resemble in many respects those just described, but the premonitory symptoms extend over a longer time, and those which mark the actual onset of the attack are less severe. The injection of the conjunctiva in this case is often confined to the circumcorneal zone, a fact which, combined with the immobility of the pupil, not unfrequently leads to a diagnosis of iritis—a mistake which may have most disastrous consequences, for, while atropine does good in iritis, it invariably does harm in primary glaucoma.

Chronic glaucoma differs from the preceding forms in the absence of conjunctival injection, and of opacities in the media. The absence of inflammatory symptoms led to these cases being formerly classed as *simple* or *non-inflammatory glaucoma*, but the distinction is probably not a sound one. Cases of chronic glaucoma often extend over many years, there being

very slight increase of tension, but progressive failure of vision, with contraction of the visual field, cupping of the optic nerve, and atrophy of its fibres.

Hæmorrhagic glaucoma is characterised by hæmorrhages from the retinal vessels in addition to the other symptoms of glaucoma.

When the media will allow of ophthalmoscopic examination, it is found that these hæmorrhages do not materially differ from those of other diseases; they appear in dark red, somewhat elongated patches, running in the direction of the retinal vessels, which they sometimes render obscure; the veins appear dilated and tortuous, the arteries are of more normal calibre; the optic disc is hazy and congested. Arterio-sclerosis is present, with or without albuminuria.

When retinal hæmorrhages exist, the other symptoms of glaucoma are less evident than in ordinary cases.

The tension is sometimes only slightly augmented. The visual field does not present the typical concentric limitation, but contains various irregular blind spots (scotomata) corresponding to the positions of the blood-extravasations; and should these be situated near the yellow-spot region, the central vision will be destroyed. Sooner or later, however, all doubt as to the nature of the case is dispelled by the onset of markedly increased tension, and of violent pains in and around the eye. In this variety operative treatment is very undesirable, as any sudden diminution of tension, such as takes place in performing an iridectomy, is liable to be followed by fresh hæmorrhage, often necessitating immediate enucleation. Should the pain, however, be so great as to demand some operative interference, sclerotomy would alone be justifiable.

Primary glaucoma usually attacks both eyes, but rarely at the same time; the affection of the second eye may set in at any time from a few hours to several years after the first.

Pathology.—As we have already seen (p. 180), the intra-ocular fluid is mainly secreted by the ciliary processes, although a small portion may be given off by the iris. Part of this fluid passes directly into the aqueous chamber; but the greater portion passes into the vitreous chamber, and from the vitreous chamber through the suspensory ligament into

the posterior part of the aqueous chamber. The *aqueous humour* thus formed flows forward from behind the iris, mainly through the aperture of the pupil, but a portion of it passes through the tissues at the periphery of the iris ; having thus reached the anterior part of the aqueous chamber, it flows between the fibres of the ligamentum pectinatum at the angle of the anterior chamber (iridic angle) and reaches the canal of Schlemm ; from this it passes into the venous plexus, situated in the vicinity of the canal ; it either enters directly into these veins by means of valvular apertures, and so enters the blood-current, or it passes into the perivascular lymph spaces surrounding the veins, and is carried by these to the capsule of Tenon. The increased tension of glaucoma is undoubtedly due to an excess of this fluid within the globe, but ophthalmologists are somewhat at variance as to the exact cause of this phenomenon. The normal intra-ocular pressure equals that of a column of mercury whose height is 25 mm. ; there is no appreciable difference between the pressure in the aqueous and vitreous chambers.

Priestley Smith believes¹ that the comparatively large size of the lens in advanced life (see p. 420) accounts for the special liability of elderly people to primary glaucoma. He found by experiment that if the vitreous chamber be overfilled with fluid, so that the lens and suspensory ligament move slightly forwards, the ciliary processes are pressed against the base of the iris, and this, in turn, against the cornea, so that the filtration channels at the angle of the anterior chamber are shut up in a manner closely resembling what is found in the early stage of primary glaucoma. He is of opinion that primary glaucoma is the consequence of a shutting up of the angle of the anterior chamber, arising precisely in this way. In the normal state of the eye the waste fluid of the vitreous body passes forwards through the suspensory ligament to mingle with the aqueous fluid ; but in glaucoma this escape of the vitreous fluid appears to be checked by closing up the space between the ciliary body and the lens, and so the vitreous chamber gets overfilled. The *immediate* cause of the obstruction appears in most cases to be a swelling up of the ciliary

¹ *Glaucoma*. London, 1879.

processes, but it is obvious that the large size of the senile lens will act as a *predisposing* cause of glaucoma wherever such swelling occurs. This opinion as to the participation of the lens is supported by the fact that swelling of the lens as the result of injury is very apt to induce glaucoma in elderly people, in whom the lens is already of large size, and less so in young people, in whom it is small. Glaucoma, however, sometimes follows cataract extraction, and this fact has been brought forward as disproving Priestley Smith's theory. Treacher Collins, however, has found, in these cases, adhesion between the cornea and lens capsule or remaining cortical substance, and so a blocking of the iridic angle. Aniridia, or supposed total congenital absence of iris, is no safeguard against glaucoma, because in all cases of this condition the base of the iris is present, and may effectually block the spaces of Fontana. Many unsuccessful iridectomies for glaucoma have been found to be due to the base of the iris still remaining adherent to the cornea.

Brailey¹ believes glaucoma to be primarily due to a vascular change; he considers that before the development of the increased tension there is always inflammation of the ciliary body, iris, and optic nerve; that this is most pronounced in the ciliary body, especially in and around its muscular fibres; that the inflamed condition gives rise in the first instance to hypersecretion of fluid from the ciliary body and iris; that the enlargement of the ciliary folds, due to their vascular turgescence, causes the advancement of the periphery of the iris towards the cornea, by which the outflow of fluid from the globe through the angle of the aqueous chamber and the canal of Schlemm is impeded.

Weber² does not believe in the theory of hypersecretion of fluid, but in a diminished outflow. He does not consider that the hindrance to the outflow is limited to the iridic angle of the anterior chamber, but that impediments may exist in the vitreous, in the suspensory ligament, the anterior chamber, the canal of Schlemm, or in the superficial layers of the sclerotic. He also is of opinion that a higher 'condition of

¹ *Lond. Ophth. Hosp. Reports*, vol. x. part ii.

² *Trans. Int. Med. Congress*, vol. iii. 1881.

albuminosity' of the intra-ocular fluid may tend to prevent its outflow. This is undoubtedly the cause in the secondary glaucoma found as one of the symptoms of cyclitis. The deepened anterior chamber is filled with an albuminous exudation, which with difficulty escapes into the canal of Schlemm.

Whatever may be the *initial cause* of primary glaucoma, whether from (i) hypersecretion of the inflamed ciliary body, (ii) impeded outflow caused by pressure of the enlarged ciliary body upon the periphery of iris, (iii) impeded outflow from the vitreous chamber by enlargement of the lens, or from all these causes combined, there are certain pathological conditions which are fairly constantly found in glaucomatous eyes. These will now be considered.

The cornea.—As mentioned on p. 427, the cornea is frequently found to be œdematous, containing small vesicles in its substantia propria.

The ciliary body.—In the *early stage* there is always inflammation of the ciliary muscle and enlargement of the ciliary folds (cyclitis). This condition of capillary distension of the ciliary body is believed by Brailey to cause increased secretion, which may of itself be sufficient to cause glaucomatous tension. Its appearance at this early stage is very similar to that of serous iritis. It differs from that affection in becoming rapidly atrophic, the atrophy being accompanied by great dilatation of the blood-vessels. In the *advanced stage* of primary glaucoma the ciliary body is always found to be atrophied; not only the muscle, but the ciliary folds, are shrunken and their vessels widely dilated. In old people this atrophy is accompanied by the formation of dense connective tissue, whilst in the more rare attacks in young people the part becomes stretched, owing to the elasticity of the tissues, thus forming a general bulging of the anterior part of the eye, and giving rise to the condition known as *buphthalmos*.

The *iris* is also slightly inflamed, and the pupil somewhat dilated and sluggish in the *early stage*. Its periphery is approximated to the back of the cornea at the iridic angle so as to diminish the size of that outlet; the fibres of the ligamentum pectinatum as they pass from Descemet's membrane

to the base of the iris are found to be swollen by hypernucleation of their epithelioid covering, and, by being thus increased in calibre, the spaces between them (spaces of Fontana) are considerably diminished, thus forming a further obstruction to the outflow of the fluid from the anterior chamber to the canal of Schlemm. In the *advanced stage* the periphery of the iris is found to be in actual contact with the cornea, and adherent to it, so that the iridic angle is more or less completely blocked. Under these circumstances the edges of the pupil are sometimes everted, and the pupillary margin of the iris no longer rests upon the capsule of the lens. Finally, the iris may become atrophied and reduced to a mere band of slate-coloured tissue around the widely dilated pupil.

The *suspensory ligament* is put upon the stretch by the accumulated intra-ocular fluid. This is probably an important factor in the impairment of accommodation which is always present in glaucoma.

The *optic nerve* is found to be somewhat inflamed in the very early stage (Brailey). In the *advanced stages* there are always changes in this structure. On transverse section the nerve-fibres are found to be shrunk, and the intervening connective tissue considerably hypertrophied. The same hypertrophy is found to affect the pial sheath of the optic nerve and the lamina cribrosa. This latter structure, which forms the floor of the optic disc, is the weakest part of the fibrous capsule of the globe, and is therefore the first to yield to glaucomatous tension. In *cupping of the optic disc* the fibres of the lamina are pushed backwards, and the nerve-fibres as they radiate towards the retina are pressed back with it. The depth of the cup depends chiefly upon the amount and duration of increased tension and the age of the patient. In a person of middle age, where the tension has been considerable and of long standing, the cup is generally deep with overhanging edges, but in an older person (over sixty) it is less deep, owing to the unyielding nature of the fibrous tissue.

The choroid.—It was formerly considered (von Graefe) that choroiditis serosa was one of the chief causes of glaucoma, but it is found that this structure is not affected in the early stage, and only evinces a tendency to atrophy in

the later period of glaucoma, when the optic disc is often seen surrounded by a ring of choroidal atrophy.

The *retina* suffers from prolonged pressure in several ways. In the first place, the compression of so delicate a structure is alone sufficient to impair its function; then we have seen that the fibres of the optic nerve are compressed and atrophied at or near the optic disc, especially as they pass over the sharp knifelike margin of the scleral ring; finally, the flow of arterial blood to the retina is impeded, and the efflux of venous blood is retarded. The want of arterial blood is probably the cause of the characteristic limitation of the visual field; the course of the vessels to the periphery being longer, and so having greater resistance to overcome than those at the centre. The vessels of the retina are frequently found to be degenerated. The walls of the arteries are often thick, and present a hyaline appearance; sometimes aneurysmal dilations may be seen, and hæmorrhages are frequently found.

Diagnosis.—The diagnosis of glaucoma is often difficult. The symptoms on which greatest stress is laid are the increase in the intra-ocular pressure, the cupping of the disc, and the typical contraction in the visual field. The first of these, however, is often intermittent; the second often absent, or invisible; and the contraction of the visual field often atypical. An acute attack of primary glaucoma may be altogether overlooked. The general condition of the patient renders a diagnosis of influenza or some acute disease apparently more obvious than that of a local lesion; the eye symptoms may be totally missed until too late for an iridectomy to do much good besides relieving the pain. A less acute attack resembles in many respects iritis, and to diagnose between the two a careful inquiry into the history of onset must be made. Premonitory symptoms are rarely absent from a subacute glaucoma. Again, the pupil is usually contracted in iritis, dilated in glaucoma. The anæsthesia of the cornea in glaucoma is often a valuable sign, as also a shallow anterior chamber; should glaucomatous cupping of the disc be seen, this is characteristic, but the media are rarely sufficiently clear for an ophthalmoscopic examination. If there is much cyclitis accompanying the iritis, further difficulty may arise, as in-

creased tension may be added to the ordinary symptoms of iritis. Chronic glaucoma is, as a rule, easy to diagnose, the optic cupping and the characteristic contracted field distinguishing it from primary optic atrophy.

Treatment.—In the year 1856 Albrecht von Graefe, having discovered that *iridectomy* was effectual in reducing intra-ocular tension, employed this operation in the treatment of glaucoma; his attempts in this direction were followed by the most brilliant success. The operation of iridectomy has been and is almost universally adopted for this disease, and is the means of rescuing hundreds of persons from blindness every year.

Before the time of von Graefe's discovery, glaucoma held a prominent place in the category of incurable diseases.

Since that time iridectomy has been universally practised, and has proved itself to be the best remedial measure at our disposal. Other operative measures, as sclerotomy and paracentesis of the aqueous and vitreous chambers, have been introduced, but have in no way been found superior to the original operation, although in certain cases their adoption may be advisable. Some surgeons combine a scleral puncture with the iridectomy.

The *method of performing iridectomy* will be found described on p. 229. When made for the relief of glaucoma, the incision should be in the sclera, close to the corneal margin; the distance between the puncture and counter-puncture should be from 6 to 8 mm. In excising the portion of iris, care should be taken to cut it off as close to its ciliary border as possible. A large iridectomy should be made involving at least one-fifth of the iris. It should, when possible, be made upwards so that the upper eyelid may afterwards cover the coloboma, and so prevent spherical aberration. It often happens, however, that the upper part of the iris is in an atrophied condition, in which case the performance of iridectomy is not only more difficult but is less satisfactory in its results, so that it is desirable to select another portion of the iris for excision. Great care must be taken to free the pillars of the coloboma from the angles of the incision, in order that the iris may not be adherent to the developing cicatrix.

Iridectomy is much more easily performed in simple non-inflammatory glaucoma than in the acute and subacute kinds; the anterior chamber is less shallow, the conjunctiva is less injected and there is no chemosis, the cornea is clear, and the iris uninflamed.

There is still considerable difference of opinion as to the way in which iridectomy relieves primary glaucoma. Some think that permanent filtration takes place through the scar, and aim at getting, if possible, a cystoid cicatrix by means of gentle massage. Others consider that the opening up of the iridic angle is the main object to be attained. Failures to relieve glaucoma by this operation are in all probability usually due to the ciliary border of the iris remaining *in situ*, in contact with, or it may be adherent to, the cornea. This may, however, be to some extent counteracted by the presence of a cystoid cicatrix; but it seems impossible that filtration can take place through an ordinary fibrous scar.

The *best time for operation* is during the early stage of the disease; whether the case be one of simple, subacute, or acute glaucoma, the earlier it is operated upon after the actual onset of the disease the greater are the probabilities of immediate and of permanent success. In *acute glaucoma* the danger in delaying an operation is so great that an eye may be sufficiently destroyed in the course of twenty-four hours to render it quite blind and beyond possibility of recovery. Should it be found necessary to postpone operation for a limited period, the danger from tension may be modified by the free use of eserine drops (F. 16) instilled every hour, combined with leeching and hot fomentations, or ice compresses. Free opening of the bowels should be secured by the use of saline purges. A hypodermic injection of morphine is often useful, since it lowers the blood pressure, diminishes the secretion from the ciliary region, and allays the pain.

In *subacute cases* the same rule applies: eserine instillation should be commenced at once, and iridectomy performed at as early a date as possible.

In *simple glaucoma* (chronic) the immediate performance of the operation is less imperative, although, from what has

been said, its beneficial results will be greater in recent than in old-standing cases.

It generally happens that patients who present themselves with acute or subacute glaucoma have previously been the subjects of the simple or chronic form of this disease, although they were possibly unaware of the fact. A careful inquiry into the history of such cases will generally elicit confirmatory evidence of this. In the treatment and prognosis, therefore, of *any* case, it behoves us to inquire carefully as to the particular stage at which the disease may have arrived, and to what extent the retina may have suffered from intra-ocular pressure; for whenever increase of tension has existed for any length of time, we find that atrophic changes have appeared in the optic disc and retina which cannot possibly be removed by reducing the tension of the eye.

The best results from iridectomy are obtained in the acute and subacute cases of recent date. Pain is speedily relieved, chemosis, haziness of the cornea, and of the other media, are quickly dispersed; vision is forthwith improved, and in the majority of cases the visual acuteness and the visual field after operation are restored to a condition nearly equal to that which existed before the onset of the inflammatory attack. This, of course, applies only to those cases which have been operated upon within a reasonable time after the onset of the attack. Exceptions, however, sometimes occur in which a well-performed iridectomy fails to reduce tension in a satisfactory manner. In such cases recourse must be had to sclerotomy or to a second iridectomy. In other exceptional cases it unfortunately happens that all operative interference fails to arrest the course of the disease, which steadily progresses to complete blindness.

In simple glaucoma little more can be effected by iridectomy than a stoppage or retardation of the progress of the disease. The conditions of visual acuteness and visual field will remain about the same as before operation, though some astigmatism often results.

The following complications may follow iridectomy for glaucoma: hæmorrhage into the vitreous chamber, either immediately or a few days after the operation; injury to the

lens, with resulting cataract ; forward displacement of the lens, with consequent continuance of the glaucoma ; prolapse of the lens through the operation wound.

The instillation of *eserine* (F. 16) or *pilocarpine* (F. 22) is of great service in all cases where for any reason operative interference is postponed. They act upon the sphincter pupillæ, which contracts and so draws the iris away from the cornea. The sulphate of eserine is about twenty-four times as strong as the hydrochlorate of pilocarpine. The constant use of either of these drugs is, however, very irritating to the conjunctiva, and a typical follicular conjunctivitis may result ; and although the disease is kept somewhat in abeyance, it will nevertheless make such advances as to render iridectomy finally compulsory. Cocaine is often advantageously used in combination with either of these drugs, since it acts as a vaso-constrictor, and also helps to allay the pain. Its dilator action on the pupil is counteracted by the eserine or pilocarpine.

In *hæmorrhagic glaucoma*, when the condition of the media will admit of the detection of hæmorrhages in the retina, the vitreous, or the anterior chamber, iridectomy is contra-indicated. Owing to the diseased condition of the blood-vessels, an iridectomy is certain to aggravate the mischief, the sudden lowering of the intra-ocular pressure causing further hæmorrhage. The operation of sclerotomy, in which the diminution of tension is more gradual, has not so far been followed by these untoward results.

In very old people the probable duration of life must be considered ; where the disease is of the mild and chronic form the central vision may continue fairly good until the end.

In advanced disease, where the vision is perhaps totally gone from one eye, and greatly impaired in the other, the iris is usually atrophied, and sclerotomy would be more easily performed than iridectomy, although but little benefit, beyond the relief of tension and consequent cessation of pain, could be expected.

Sclerotomy has of late years been extensively tried as a substitute for iridectomy in glaucoma. Various methods of performing sclerotomy are in use ; the following plan, as recommended by de Wecker, is the one I usually adopt.

Operation.—A Graefe's linear knife (fig. 87, p. 393), or one of de Wecker's sclerotomes, is introduced into the sclerotic at 1 to 2 mm. from the margin of the cornea, slightly more posterior than in an iridectomy for glaucoma; its point is carried across the anterior chamber in front of the iris, and the counter-puncture made in the opposite corresponding position. The knife is now carried upwards by a sawing movement until its edge is just covered by the sclero-corneal junction, that is, until its edge forms a tangent with the highest point of the cornea; the incision is then stopped without cutting through the remaining bridge of sclerotic above, and

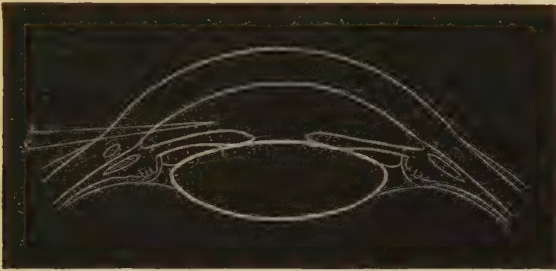


FIG. 96.—Lines of Incision.

I, in iridectomy; *S*, in sclerotomy.

the knife is slowly withdrawn. Great care should be taken not to wound the iris, also to prevent a sudden rush of the aqueous from the wound, whereby the iris might be caused to protrude, and so become entangled in the wound.

By sclerotomy performed in this manner all the tissues at the iridic angle are divided, except the bridge of sclerotic tissue which is left. The operation is 'subconjunctival.' The line of incision which it is desirable to obtain in sclerotomy is shown in fig. 96, where it is seen to be somewhat posterior to that of iridectomy. It is evident that in this operation, if the incision is too far removed from the cornea, there is danger of wounding the ciliary body, and consequent hæmorrhage into the vitreous chamber, also of possible plastic cyclitis, and consequent sympathetic inflammation in the other eye.

With the view of preventing the prolapse of the iris through the wound in the sclerotic, as well as for the continued reduction of tension, the use of eserine (F. 16) is advisable both before and after the operation.

Sclerotomy, although admitted by some to be theoretically equivalent to iridectomy, has not yet gained the universal confidence of ophthalmic surgeons. Speaking on this subject in 1878, de Wecker,¹ one of the strongest supporters of sclerotomy, says: 'Although I shall, probably, during the whole course of my career continue to give preference to excision of the iris as being the surest operation against glaucoma, I hold the conviction that our progressive science will substitute for it a simpler and more logical proceeding. . . . Under two circumstances only do I strongly recommend you to renounce iridectomy and to resort to my operative procedure; first, when you recognise that you are dealing with hæmorrhagic glaucoma, for here the double section with the narrow sclerotome (2 mm.) enables you to avoid the danger of the section for iridectomy; and secondly, in cases of absolute glaucoma: in these, sclerotomy ought always to be preferred to iridectomy, the operation being undertaken only with the object of freeing the patient from severe pain.'

Paracentesis of the vitreous chamber is practised by Cowell for the relief of certain forms of chronic and secondary glaucoma; he plunges the point of a Beer's cataract knife to the extent of 5 mm. through the conjunctiva, sclerotic, choroid, and retina, as near as possible to the space between the insertions of the superior and external recti. The incision is sometimes attended with excellent results, not only in relieving tension, but in the improvement of vision, &c.

Resection of the cervical sympathetic has been performed during the last few years by Tonnesco, Abadie, and others. These surgeons contend that this operation is especially indicated in cases of chronic glaucoma. The superior cervical ganglion is removed, and there appears to be little doubt that decreased intra-ocular tension is one of the results of this procedure. It is, however, doubtful whether this is permanent, and it is accompanied by severe constitutional symptoms.

When an eye is lost from glaucoma and continues to be painful after sclerotomy or any other operation, excision of the globe is the only remedy.

¹ *Thérapeutique Oculaire*, part i. p. 378. 1878.

SECONDARY GLAUCOMA.

Secondary glaucoma signifies a condition of increased intra-ocular tension, occurring as a complication of some other affection of the eye. It is most common amongst those maladies which interfere with the normal movements and position of the iris.

Etiology.—*Perforating ulcer or wound of the cornea with protrusion of the iris* is a common example of this. The whole or part of the pupillary edge of the iris becomes entangled in the wound, where it appears as a black point; if this be carefully examined, the protruding portion of the iris will be found to act as a filter, and for a certain period to give rise to a constant leakage; finally, this black point becomes covered over by a layer of lymph which cicatrises and the leak is closed. Increased tension is the immediate result; the fluid can no longer pass through the cicatrix; the periphery of the iris is jammed against the cornea by the fluid pressure behind it, and so the entrance to the canal of Schlemm is closed. Unless the tension is relieved by iridectomy, or an equivalent operation, the iris becomes atrophied and adherent to the cornea; anterior staphyloma or bulging of the whole anterior part of the eye may occur; the iris is greatly stretched, and tension is made upon the ciliary processes; the lens is carried forward as well as the iris; the zonula is stretched, and so traction is made upon the pars ciliaris retinæ. The vitreous undergoes degeneration, and becomes more fluid than normal.

Complete posterior synechia and *annular posterior synechia* are common causes of secondary glaucoma: the pupil being bound to the anterior capsule of the lens, the passage of fluid forwards through this is arrested; pressure is thus made upon the iris from behind, and its peripheral portion is bulged forwards, thus closing the angle of the anterior chamber, while the attachment of its pupillary edge to the lens gives a funnel-shaped appearance to the pupil, the condition being termed *iris bombé*.

Wound of the lens, as in the needle operation or by accident, often gives rise to increased tension, probably by causing

swelling of the lens-structure within its capsule. The remains of soft lens matter after cataract extraction is also a cause of increased tension.

Cataract extraction may be followed by glaucoma. This may be due to one of three conditions: an intervening iritis may produce exclusion or occlusion of the pupil; some remaining soft lens matter may become adherent to the scar, and fill up the coloboma iridis; lastly, an implantation cyst may line the whole of the anterior chamber, some epithelium being carried in by the knife and subsequently proliferating.

Dislocation of the lens either forwards or backwards, and partial dislocation of the lens, sometimes give rise to glaucoma.

Foreign bodies in the globe may cause glaucomatous tension; this, again, is probably generally by wound of the lens and iris or ciliary body.

Sarcoma of the choroid usually, and *glioma of the retina* occasionally, are attended at some period of their history by a rise of tension. This sign occurring in an eye with retinal detachment is strong evidence in favour of an intra-ocular new-growth.

Cyclitis, or *serous iritis*, is usually accompanied by increased tension in its earlier stages.

Symptoms.—Increased tension is the chief sign of secondary glaucoma. The other symptoms of contracted field, haloes, impaired visual acuity, and changes in the refractive condition, are seldom to be made out, on account of the lesions of the cornea, iris, lens, &c. As in primary glaucoma, the anterior chamber is usually shallow. Where, however, the glaucoma is secondary to cyclitis, the chamber is abnormally deep. This is because the increased pressure is due, not to a blocking of the iridic angle, which is abnormally open, but to the increased albuminosity of the aqueous fluid.

The treatment of secondary glaucoma must vary with the cause. In the case of a dislocated lens being the cause of the trouble, an attempt should be made to remove it (see p. 413). Where the iris is adherent, either anteriorly or posteriorly, iridectomy (see p. 229) should be performed. It is often of great importance, though at the same time extremely difficult, to determine whether in secondary glaucoma due to iridic

adhesions or to active irido-cyclitis, atropine or eserine should be used. As a general rule, it may be said that atropine is indicated, but the case must be carefully watched. Iridic adhesions may in this way be broken down, and so the path through the pupil opened up; active inflammation may be allayed with a return to a more fluid aqueous. When the eye is quite blind and the media opaque, if it is painful, and especially if the other eye remains unaffected, it probably contains a tumour, and should therefore be excised.

The *after-treatment* of iridectomy or sclerotomy for glaucoma is simple enough. After iridectomy, the eyelids are closed and covered with a piece of dry aseptic gamgee tissue and a light bandage, or adhesive plaster. After sclerotomy, the bandage is generally dispensed with altogether, and the eyes shaded from the light. On the second or third day the use of eserine (F. 16) should be resumed. The general health should be supported by tonics, any excess in diet, and especially in alcoholic drinks, being avoided. Any constitutional dyscrasia, as gout or rheumatism, should be combated by suitable remedies. For the first few days the patient had better be kept in bed, and should not leave the darkened room for at least a week. After that time he should wear spectacles of the darkest neutral tint; he should avoid over-use of the eyes, and be as far as possible removed from over-work and worry.

CONGENITAL GLAUCOMA, BUPHTHALMOS, OR HYDROPHTHALMOS.

This condition, popularly termed 'ox-eyed,' is by no means rare. As in all forms of glaucoma, obstruction at the iridic angle is probably the pathological cause, the obstruction being the result either of intra-uterine iritis or of the incomplete developmental separation of the anterior layers of the iris from the posterior layers of the cornea. Owing to the great elasticity of the coats of the eyeball in early life, a general enlargement of the globe takes place. The diameters of the cornea may be double their normal size; the cornea is considerably thinned. The anterior chamber is, as a rule, very deep, and iridodonesis is often present. The optic nerve is

cupped. Subsequently, the cornea becomes opaque, the lens dislocated, the tension raised, and total blindness ensues. In a few cases the disease becomes stationary, and sight is not altogether lost. This is probably due to the obstruction giving way, leading to the normal exit of the intra-ocular fluid. The treatment is eminently unsatisfactory. In the late stages where the patient is totally blind, evisceration or enucleation is indicated, though chiefly for cosmetic purposes. When the disease is seen quite early where the tension is normal or only slightly raised, iridectomy should be performed. In intermediate stages, sclerotomy or repeated paracentesis is perhaps the better treatment, iridectomy being in these cases usually followed by profuse intra-ocular hæmorrhage and cyclitis.

CHAPTER XIV.

THE REFRACTION OF THE EYE.

By JOHN GRIFFITH and HENRY JULER.

I. OPTICAL PRINCIPLES—II. THE EYE CONSIDERED AS AN OPTICAL INSTRUMENT—III. NORMAL REFRACTION, OR EMMETROPIA—IV. ERRORS OF REFRACTION, OR AMETROPIA—V. LENSES—THE OPHTHALMOSCOPE—VI. METHODS OF ESTIMATING REFRACTION—VII. GENERAL CONSIDERATIONS.

Section I.—OPTICAL PRINCIPLES.

FROM every point on the surface of an illuminated or luminous object light is given off in every direction in straight lines. Hence the light from any such point may be considered, and represented diagrammatically, as fine radiating lines; such imaginary lines are called *rays*. Adjacent rays coming from the same point constitute a *pencil* of light. Rays of light coming from any one point must necessarily be *divergent*: the greater, however, the distance of the source of the light, the more nearly will they approach to parallelism. Thus, in the case of rays from the sun, it is impossible by the most accurate measurement to demonstrate that they are not parallel. Here we shall be chiefly concerned with rays which enter the eye through the pupil; and of these we may, for all practical purposes, consider as parallel those which come from a point distant not less than 6 mètres (20 feet). Convergent rays do not exist in nature.

As long as rays of light travel in the same medium they continue their original course; if, however, they pass into a medium of different density, they undergo a change of direction; some are reflected, some are refracted, and some absorbed.

Reflection and refraction occur at the surface which separates the two media.

A. Reflection.

Reflection may occur at almost any surface, but it is greatest from the highly polished surface of an opaque body. The laws of reflection are as follows :

(a) *The plane of reflection coincides with the plane of incidence.*

If any ray of light falls on a reflecting surface, it is reflected in the same vertical plane, but not along the path of incidence unless its original direction is perpendicular to the reflecting surface.

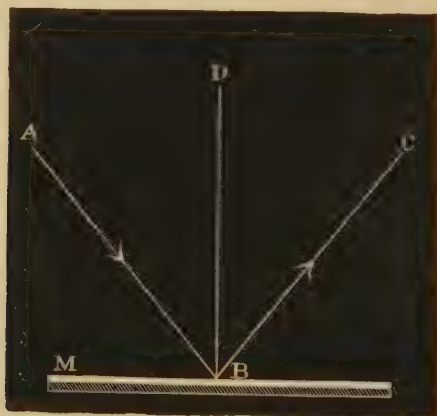


FIG. 97.—Reflection by a Plane Surface.

(b) *The angle of reflection is equal to the angle of incidence.*

The angles of incidence and reflection are the angles which the incident and reflected rays respectively make with the normal or perpendicular to the reflecting surface.

In fig. 97 let *M* be a plane mirror and *A B* an incident ray which strikes the reflecting surface at *B*: it will be reflected to *c*. Draw *B D* perpendicular to the surface of the mirror. The angle of reflection *D B C* is equal to the angle of incidence *D B A*.

Reflection by a Plane Mirror.—The reflection of an object

in a plane mirror is seen as a virtual erect image situated behind the mirror.

The image and the object are equidistant from the mirror, and are always of the same size.

Assuming the object to be a lamp, the reflected rays would appear to come from a virtual image at an equal distance behind the mirror.

In fig. 98 let M be a mirror and L a light, the observer O will see L as if it came from L' , its virtual image. L and L' are equidistant from the mirror M .

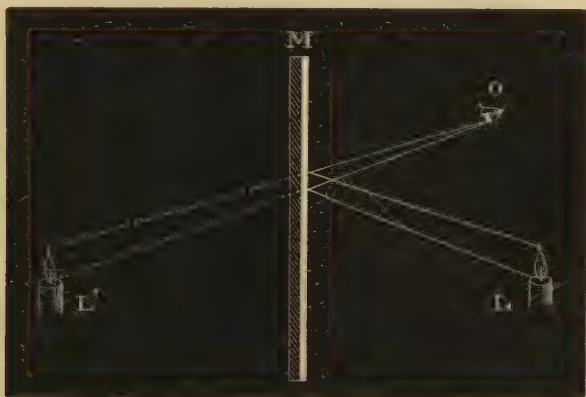


FIG. 98.—Reflection by a Plane Mirror.

If a plane mirror be rotated on any axis in its own plane, the virtual image of the object moves in an opposite direction to the rotation of the mirror.

In fig. 99 let M be a mirror, O an observer, and AB an object of which ab is the (virtual) image. If the mirror M be rotated upwards on its horizontal axis to M' , ab will move downwards towards $a'b'$ in order to maintain its proper relationship to the mirror according to the laws of reflection. The observer will see the reflection from a different portion of the mirror.

Reflection by a Concave Mirror.—In a plane mirror the reflected image was seen to be always virtual and erect. The image and object appeared equidistant from the mirror, but

on opposite sides, and always of the same size as one another. In a concave mirror, however, three different phenomena may occur according to the relationship the object bears to the principal focus of the mirror.

If the object be placed at the principal focus, the reflected rays are parallel, so that a blurred reflection without any image is seen.

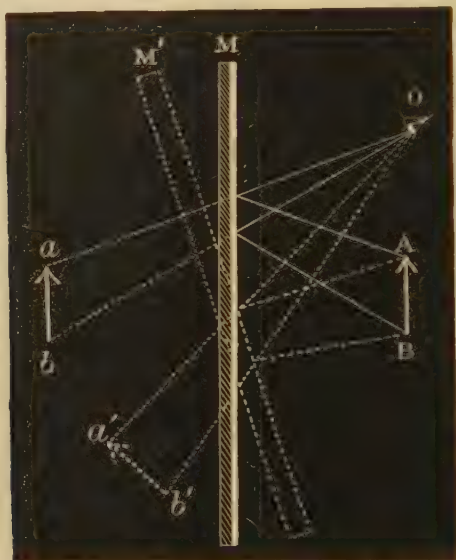


FIG. 99.—Movement of the Virtual Image by Rotation of the Mirror.

If the object be placed within the principal focus, an erect enlarged virtual image of the object is visible, which diminishes in size as the object approaches the mirror.

*If the object be placed beyond the principal focus, the reflected rays are convergent, and come to a focus in front of the mirror, forming a real inverted image of the object. The image is the same size as the object if the latter is situated at the centre of curvature; larger if the object is in front, *i.e.* between the centre of curvature and the principal focus; smaller if beyond the centre of curvature.*

To understand these phenomena it will be necessary to explain the reflection of simple rays from a concave mirror.

In a plane mirror all rays parallel to any given perpendicular ray are themselves normals to the reflecting surface.

In spherical mirrors all rays parallel to the principal axis are brought, directly or indirectly, to a real or virtual focus; *i.e.* they all meet in a point.

The principal axis of a spherical mirror is an imaginary line drawn through the centre of the mirror and passing through its centre of curvature.

All radii of curvature are themselves normals, *i.e.* axes of incidence. Spherical mirrors, therefore, differ from plane mirrors only in that each point has its own axis of incidence.

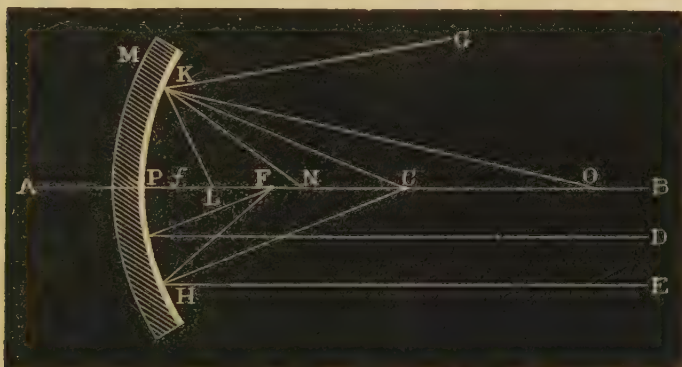


FIG. 100.—Reflection by a Concave Mirror.

In fig. 100 let M be a concave mirror, AB its principal axis, and C its centre of curvature; any rays, D and E , parallel to AB will meet after reflection on AB at F . F is, therefore, the principal focus of the mirror, and f its focal length, which is half the radius of curvature CP . To the point H at which the parallel ray EH strikes M draw the radius of curvature CH , the angle CHF is equal to the angle CHE . Since all parallel rays come to a focus at F , all rays from F are reflected parallel. Rays from any point within the principal focus are reflected divergent. Rays from any point beyond the principal focus are reflected convergent. Let LK be a ray coming from any point L within the principal focus F , and striking M at K ; it will be reflected towards G , diverging from the principal axis. Let NK be a ray from any point N on the principal axis between

r and c ; it will on reflection meet the principal axis at o beyond the centre of curvature c . Conversely, any ray from o will meet AB at n after reflection. That is to say, all rays from n would, after reflection, come to a focus at o , and all rays from o would come to a focus at n ; o and n are conjugate foci.

If the object be placed within the principal focus, a virtual erect image is formed behind the mirror.

In fig. 101 let OP be an object within the principal focus F ; rays from OP will be reflected divergent as if coming from a virtual image behind the mirror. From the centre of curvature c draw the axis of incidence co , passing through the extremity of the object OP . From o draw another ray oH ; it will, according to the laws of reflection, be reflected towards k .

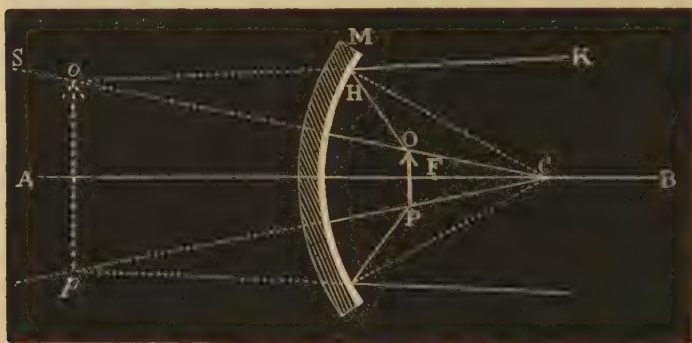


FIG. 101.—Virtual Erect Image formed behind a Concave Mirror.

The virtual image of the point o of the object will be situated at the point o , where $κH$ (produced) meets cs ; and all rays from o will, after reflection, appear to proceed from that point. Similarly, the point P will have its virtual image at p ; op is, then, the erect virtual image of the object OP .

If the object be situated beyond the principal focus, a real inverted image is formed in front of the mirror.

In fig. 102 let OP be an object beyond the centre of curvature c ; rays from the object OP will be reflected convergent, and will form a real inverted image between c and the principal focus F .

From the point o , the extremity of the object OP , draw the ray oc passing through the centre of curvature; it will

be reflected back along the same path, since it is an axis of incidence. From o draw another ray oD parallel to the principal axis AB ; it will, on reflection, pass through the principal focus F and meet the axis of incidence CG at o ; all other rays from o would meet at o ; therefore o is the image of o . Similarly p can be shown to be the image of p ; and since all other points between o and p of the object op have corresponding images between o and p , op is the real inverted image of op .

Let it be supposed that op is the object, its image would be op ; i.e. the corresponding points which build up the object op and the image op are conjugate foci.

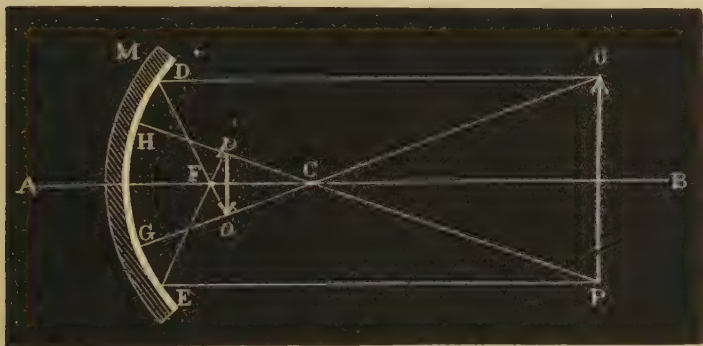


FIG. 102.—Real Inverted Image formed in front of a Concave Mirror.

If a concave mirror be rotated, the real inverted aerial image of any object will move in the same direction as the rotation of that mirror.

For example, if a concave mirror be held 1.5 m. (5 ft.) from a lamp, an inverted image of that lamp will be formed just beyond the principal focus of the mirror; it will move upwards on upward rotation of the mirror, and *vice versa*.

Let the real inverted image op of the object op (fig. 103) on the principal axis AB be situated just beyond the principal focus of the mirror. If the mirror be rotated upwards op will move upwards. Rotate M to M' ; the object is no longer on the principal axis but below it, for AB has moved to $A'B'$; consequently, by the laws of reflection, the image will be

formed above $A' B'$ at $o' p'$; thus, the image has moved upwards in the same direction as the rotation of the mirror.



FIG. 103.—Movement of the True Image by Rotation of the Mirror.

Reflection by a Convex Mirror.—*The reflection of an object by a convex mirror is seen as a small virtual and erect image behind the mirror.*

Parallel rays falling on a convex mirror are reflected divergent as if coming from a point behind the mirror. This point is called the *virtual principal focus* of the mirror.

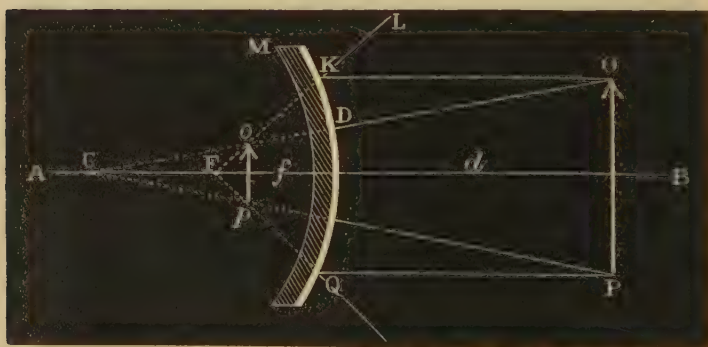


FIG. 104.—Reflection by a Convex Mirror.

Divergent rays are reflected more divergent than parallel rays, and, being prolonged backwards, meet at a point within the principal focus of the mirror.

Thus only distant objects can form virtual images at the

principal focus; the virtual images of near objects are formed within it.

In fig. 104 let m be a convex mirror, c its centre of curvature, F its principal focus, and f its focal length. Let $o p$ be any object. From the centre c draw $c o$, a prolonged radius of curvature, meeting the extremity of the object $o p$. Since $o p$ coincides with the radius of curvature $c p$, it is an axis of incidence; so the reflected ray passes back along the path of incidence.

From o draw $o k$ parallel to $A B$; it will be reflected towards L . Prolong $L k$ backwards, it will meet the principal axis at the principal focus, since $k L$ is a reflection of a parallel ray. At the point o at which $L F$ crosses $o c$, all reflected rays of the point o will meet if prolonged backwards; o is the virtual image of the point o of the object. In a similar manner, p may be shown to be the virtual image of the point p . Therefore, $o p$ is the virtual erect image of the object $o p$.

The size of the image will be to the size of the object as the focal length f of the mirror is to the distance d of the object from the centre of curvature. That is $\frac{i}{o} = \frac{f}{d}$ (i = image, o = object). Writing r for the radius of curvature, since $r = 2f$ we have $\frac{i}{o} = \frac{r}{2d}$ or, expressed differently, $r = \frac{2di}{o}$.

Any three of these terms being known, then, we can by the above formula find the fourth.

B. Refraction.

When rays of light pass obliquely from one medium into another of different density, those that are not reflected upon entering the new medium undergo a change of direction; that is to say, they are turned aside out of their original course. They continue their new direction until they meet with another medium, when they will again suffer deflection. This change in the ray's course is called *refraction*. The direction and

amount of the change depend on two factors—the difference in the refracting power of the two media, and the form of the surface of separation. As a rule, the refracting power of a medium is in proportion to its density; thus glass is more refracting than water, and water than air, while air, as compared with vacuum, has a definite power of refraction. The following are the laws of refraction: (1) *A ray in passing from a less into a more refracting medium is refracted towards the normal.* (2) *In passing from a more into a less refracting medium a ray is refracted away from the normal.* A ray whose course coincides with the normal undergoes no change of direction. The normal, in the case of a plane surface, is the perpendicular to the surface drawn from the point of contact of the ray; in the case of a spherical surface it is identical with the radius of curvature.

It is evident that if a ray pass right through the more refracting medium into the same medium which it traversed before, it will be refracted at the surfaces both of entry and of exit.

Refraction at a Plane Surface.—We will consider first the case of a ray passing through a piece of glass *whose surfaces are parallel*, as in an ordinary plate-glass window. Let $a b$ (fig. 105) be such a ray passing through n ; then at h it is refracted towards the prolongation of the perpendicular $p h$, and at h' it is again refracted away from the perpendicular $p' h'$ to the same extent—*i.e.* the amount of refraction is the same, but its direction is opposite to that which it underwent on entering the glass; hence its ultimate direction is parallel to that which it originally had, and it has merely undergone *parallel displacement*. The amount of this displacement obviously depends on the obliquity with which the ray strikes the glass, and on the thickness of the latter. In all the cases with which we shall be concerned the parallel displacement may be disregarded, and rays which pass through a body whose surfaces are parallel or concentric may be considered to be unchanged in their course, provided that the media on each side of the body have the same power of refraction.

If, instead of being parallel, *the surfaces of the glass converge as in a prism* (fig. 106), the prolongations of the normals,

$p h$ and $p' h'$, are no longer parallel, but are directed towards the base of the prism; hence the ray $a b$, following the laws of refraction, will also be refracted towards the base both on entering and leaving the glass. Therefore, *rays passing through a prism are refracted towards its base.*

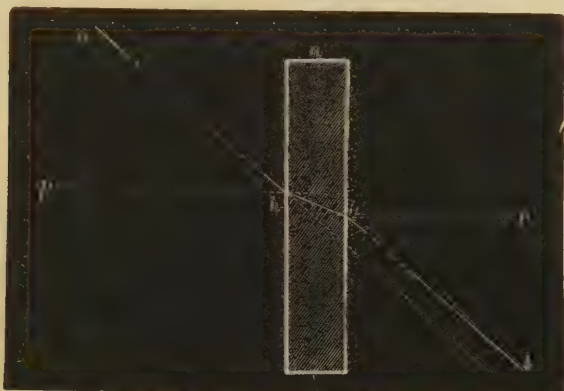


FIG. 105.—Refraction through a Medium with Parallel Surfaces.

By refraction at a plane surface the *actual* direction of rays is changed, but not their *relative* direction; thus rays which were parallel or divergent before remain parallel or divergent



FIG. 106.—Refraction through a Prism.

after refraction. This results from the fact that the normals to a plane surface are parallel to each other.

Refraction at a Spherical Surface.—If the separating surface is curved (fig. 107), the normals are no longer parallel, but, in the case of a spherical surface, meet at the centre of curvature; hence rays by refraction at such a surface are rendered

more or less divergent, according as they are made to approach or recede from the normals.

We will consider first refraction at a single spherical surface.

Let cd (fig. 107) be a portion of such a surface separating the media n and n' , of which n' is the more refracting, and let k be its centre of curvature. Then lines drawn from k to any points in cd will constitute normals to the surface cd , and, since rays which coincide with the normal are unrefracted, any ray which is directed to k is unrefracted—hence k is called the *optical centre*—(it coincides in this case with

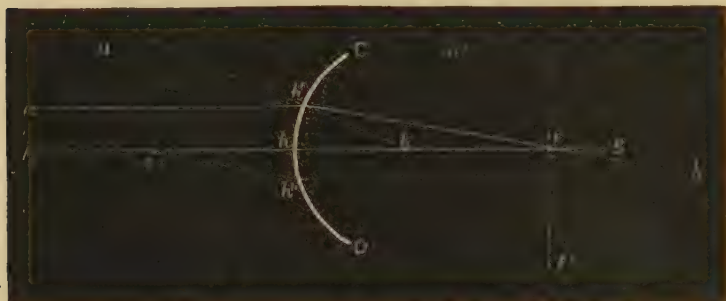


FIG. 107.—Refraction at a Single Spherical Surface.

the centre of curvature). A line, AB , joining in the centre of cd (h) with k , is called the *principal axis* of the surface; all other rays which pass through k are called *secondary axes*.

Let ah' be a ray parallel in n to the principal axis AB . Draw the normal $h'k$; then at the point h' the ray ah' will be refracted towards $h'k$, and would intersect the principal axis AB at F' . In the same way any other ray parallel in n to the principal axis would intersect it at F' . The point at which the rays of a pencil meet after refraction is called a *focus*. The focus for parallel rays is called the *principal focus*. The distance (hF') of the principal focus from the refracting surface is called the *principal focal distance*. Rays parallel to any secondary axis are focussed on that axis in the same vertical plane as the principal focus; this plane ($F'F''$) is called the *principal focal plane*.¹

¹ It would be more accurately represented by the arc of a circle having k as centre, and kF' as radius.

The radius of curvature (r) of the refracting surface, and the relative refracting power of the two media (n and n'), being known, the principal focal distance F can be found by the formula—

$$(1) \quad . \quad . \quad . \quad . \quad . \quad F = \frac{n' r}{n' - n}.$$

Rays coming from n' and passing into n are of course subject to the same laws, so that rays which are parallel to the principal axis in n' will have their focus on it in n ; this is called the *anterior focus* (F^a). Its distance from the refracting surface can be found by the formula—

$$(1 a) \quad . \quad . \quad . \quad . \quad F^a = \frac{n r}{n' - n}.$$

Now let a ray $f h'$ (fig. 108), instead of being parallel to the principal axis $A B$, come from some point, f , on it; since $f h'$ diverges from $A B$, meets the normal $h' k$ at a greater angle than if it were parallel to $A B$, so that the same change in its direction will not cause it to intersect $A B$ at F , but at some greater distance, f' ; and any other rays from f would meet at f' . Conversely, if we considered the rays as starting from f' they would be focussed at f , hence the two points f and f' are said to be *conjugate foci*. Conjugate foci are situated on the same axis. The principal focal distance (F) being known, the conjugate focus (f') of any point (f) can be found by the formula—

$$(2) \quad . \quad . \quad . \quad . \quad . \quad \frac{1}{F} = \frac{1}{f} + \frac{1}{f'},$$

provided that the foci are on *opposite* sides of the refracting surface; the case in which they are both on one side will be considered presently.

If the positions of k and F are known, the conjugate focus of f can also be found by construction. Draw $h k$ (fig. 108) parallel to $f h'$; prolong it to meet the principal focal plane at F' ; then $h F'$ forms a secondary axis. Since $f h'$ is parallel in n to the secondary axis, $h F'$, it will after refraction intersect the latter at the principal focal plane $F F''$, *i.e.* at F' , but the conjugate focus of f must lie on $A B$; prolong $h' F'$ until it intersects $A B$, and the point of intersection, f' , will be the conjugate focus of f .

Both from the formula (2) and from fig. 108 it is evident that the nearer f is brought to the refracting surface the farther will f' recede; when the distance of f from the surface

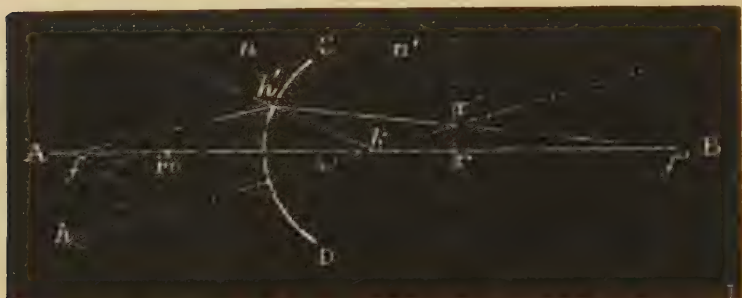


FIG. 108.—Conjugate Foci on opposite sides of the Refracting Surface.

is equal to twice the principal focal distance ($f = 2F$), f' will be at the same distance on the other side; so we get this rule:

When conjugate foci are at equal distances from the refracting surface, that distance is double that of the principal focus.

If f coincides with F^a , the rays in n' will be parallel; if it is brought still nearer to the refracting surface, as in



FIG. 109.—Conjugate Foci on the same side of the Refracting Surface.

fig. 109, the rays will diverge in n' , and therefore would meet only if prolonged backwards, so that the conjugate focus of f would now be on the same side of the refracting surface (f' , fig. 109).

The conjugate focus of f is now said to be *negative*, and is a *virtual*, as distinguished from a *real*, focus—i.e. it is not formed by a meeting of the actual rays, but of their imaginary

prolongations ; and formula (2) must now be altered by giving the minus sign to f' , so that it becomes :

$$(2\ a) \quad \dots \quad \frac{1}{F} = \frac{1}{f} - \frac{1}{f'}$$

So far we have considered refraction at one spherical surface only ; if, however, a ray passes through the more refracting medium, and again emerges into the less refracting, it is refracted again at the second surface.

Lenses are portions of a highly refracting substance, generally glass, having one or both surfaces curved. Those with which we shall deal at present are biconvex and biconcave, and their surfaces are portions of a sphere ; they are therefore called spherical lenses ; later on we shall have to deal with cylindrical lenses.

A biconvex lens renders rays less divergent, and a biconcave renders them more divergent, at both surfaces.

If parallel before refraction, the convex lens will render them convergent, and the concave divergent.

The above rule as to the action of lenses only applies if, as is usually the case, the material of the lens is more refracting than the medium in which it is placed ; if these conditions are reversed, the convex lens becomes a diverging and the concave a converging lens. Divers sometimes use spectacles in which the lenses are formed of air, *i.e.* they are composed of two curved plates of glass enclosing a cavity which is the shape of a concave lens, and contains air. A concave air-lens of this nature, when used in water, has the same effect as a convex glass-lens in air. In air it would have no action.

In a bispherical lens, the *principal axis* is the line joining the centres of curvature of the two surfaces ($c\ c'$, fig. 110).

In considering refraction at a single surface, we saw that rays which passed through the optical centre (which, in that case, coincided with the centre of curvature) underwent no change of direction ; in double refraction, the only ray whose course remains absolutely unchanged is the one which coincides with the principal axis ; for every bispherical lens, however, there are two '*nodal points*' ($k^1\ k^2$, fig. 110), whose relation to each other is such that a ray which is directed to the one before refraction is directed to the other after refraction,

and its course is then parallel to its previous direction. The ray $a b$ (fig. 110) therefore undergoes parallel displacement; it is evident, however, that, except in very thick lenses, or with great obliquity of the incident ray, a very trifling difference would be made by drawing the ray through a point o between the nodal points. Such a point constitutes the *optical centre*, and rays which pass through it may, for all practical purposes, be considered to undergo no change in their direction, and to constitute *secondary axes*.

The principal focus of a bispherical lens is found by the following formula, r being the radius of the first surface, and

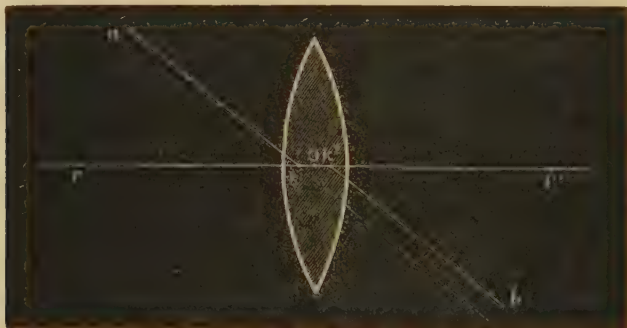


FIG. 110.—Axes of a Bispherical Lens.

r' that of the second, and n' and n the refractive indices¹ of the material of which the lens is made, and of the medium in which it is placed, respectively :

$$(3) \quad \frac{1}{F} = (n' - n) \left(\frac{1}{r} + \frac{1}{r'} \right).$$

In most bispherical lenses, the curvature of both surfaces is the same; and, as the index of refraction of glass is approximately 1.5, and that of air is 1.0, the formula becomes :

$$\frac{1}{F} = \frac{(1.5 - 1) 2}{r}; \text{ or } F = r;$$

that is to say, in bispherical lenses with similar surfaces the principal focal distance is equal to the radius of curvature.

¹ The index of refraction of any substance is its refractive power as compared with that of air, the latter being expressed by unity.

Conjugate foci are found by formulæ (2) and (2 *a*), as in single refraction.

At the conjugate focus of any point an exact image of the point is formed. When the image is formed by the actual meeting of the rays it is said to be *real*, when it is formed only by an imaginary prolongation of the rays it is said to be *virtual*.

The image of an object is the sum of the images of all points of the object. The position and size of the image can therefore be found by finding the position of the conjugate focus of the extreme points of the object. For the images of all the other points of the object will lie between these, and in the same focal plane.

As rays coming from any point on an axial ray are focussed on the same axis, and as the course of the latter is not changed,

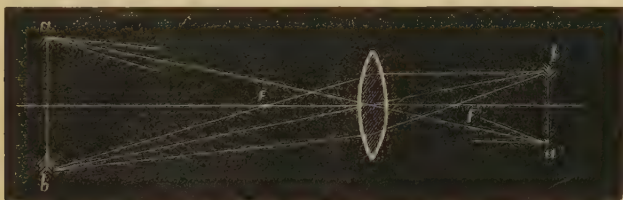


FIG. 111.—A Real Inverted Image formed by a Convex Lens.

it follows that the size of the image in relation to that of the object is the same as the relation of their distances from the optical centre.¹

Examples.—If the object (*a b*, fig. 111) be situated at more than twice the principal focal distance, the image (*b' a'*) is smaller than the object, real, and inverted.

If situated at twice the principal focal distance, it is of the same size, real, and inverted.

The farther the object from the lens the nearer will be its inverted image to the principal focus of the lens.

If the object is beyond the principal focal distance, but at less than twice that distance, the image is larger than the object, real, and inverted. This will be seen if, in fig. 111, *a' b'* is considered as the object, and *a b* as the image.

¹ Strictly speaking, as the distance of the image and the object respectively from the nodal point which is situated on the same side of the optical centre.

If situated at the principal focus (fig. 112), the rays would be parallel, and, as they would never meet, no image would be formed.



FIG. 112.—No Image is formed of an Object situated at the Principal Focus of a Convex Lens.

If nearer still (fig. 113), the rays would be divergent, and would therefore meet only when prolonged *backwards*; the



FIG. 113.—A Large Virtual Erect Image formed by a Convex Lens.

image is therefore larger than the object, virtual, and erect. Such an image could be seen only by looking *through* the lens.

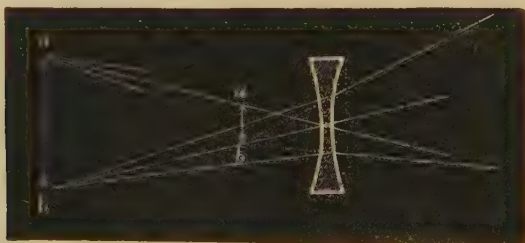


FIG. 114.—A Small Virtual Erect Image formed by a Concave Lens.

With a concave lens (fig. 114) the image is always smaller than the object, virtual, and erect.

When the image is real it can be projected on to a screen, but this cannot of course be done with virtual images, which can be seen only by looking *through* the lens.

Spherical aberration.—We have hitherto assumed that rays coming from any point are accurately focussed in a point; this is, however, only true of those which fall upon the refracting surface at no great distance from its principal axis. As long as the aperture of a lens (*i.e.* the angle formed by lines drawn from its edges to the principal focus) does not exceed 12° , the error from this source may be disregarded. But rays which fall upon the refracting surface beyond this limit are refracted more powerfully than the more central rays; this, which is called *spherical aberration*, causes slight loss of definition in an image; it can be overcome in optical instruments by the use of diaphragms, by employing refracting surfaces whose curves are parabolic, and by a combination of lenses.

Chromatic aberration.—Impaired definition of the image also arises from the fact that all the constituents of colourless light are not equally refracted; thus the red waves are the least, the violet the most refractile (see Chapter X.) If an image of a brightly illuminated white spot be formed on a screen by a lens, the central part will be white because there all the rays are combined, but the edge will be fringed with colour; this is called *chromatic aberration*, and is overcome in optical instruments by using a combination of lenses composed of different materials.

Section II.—THE EYE CONSIDERED AS AN OPTICAL INSTRUMENT.

For distinct vision three factors must be associated: (1) well-defined images of external objects must be formed on the retina at the posterior pole of the eye.

(2) The nervous elements of the retina which correspond to this image must be stimulated, and the effect be conducted to the brain.

(3) The mind must be able to interpret correctly the impressions thus received. The first is the result of the optical properties of the eye, and with it alone we are here concerned.

The eye is a closed, nearly spherical, chamber, measuring 22.15 mm. in its antero-posterior diameter. It is almost impervious to light except in front, where it is closed in by a transparent membrane, the cornea, which is more sharply

curved than the opaque portion of the investing tunic, having a radius of curvature of nearly 8 mm.

The opaque portion of the sphere is formed by a firm fibrous membrane, the sclerotic, whose structure is continuous with that of the cornea. This is lined by an extremely vascular membrane, the choroid, and this again by a layer of nervous tissue, the retina, which is an expansion of the optic nerve. The latter enters the eye a little to the inner side of its posterior pole through an aperture in the sclerotic and choroid.

The eye contains a transparent fluid, the aqueous humour, and a transparent gelatinous substance, the vitreous; the refractive indices of these are, however, almost the same, and for optical purposes they may be considered as a single medium, having an index of refraction of 1.337. The refractive index of the crystalline lens diminishes from the nucleus outwards; its mean value may be taken to be 1.4545.

Since the surfaces of the cornea are parallel, rays passing through it alone, from air on the one side into air on the other, would merely undergo parallel displacement. Its thickness may therefore be disregarded, and it may be looked upon as the surface of separation between the air and the intra-ocular fluids. If this constituted the whole of the refracting system of the eye, as it does after the operation of cataract extraction, its principal focal distance calculated by formula (1) would be about 31.5 mm.; but suspended in the eye, between the aqueous and the vitreous, is a biconvex lens of still more highly refracting substance; this is placed in the eye in such a position that its optical centre is 5.8 mm. behind the anterior surface of the cornea. The effect of this combination is such that the principal focus for the whole eye is 22.15 mm. from the cornea, that is, on the retina. The following are the optical constants of the normal eye which are the most important (Helmholtz):

	mm.
Radius of curvature of cornea	8
„ anterior surface of lens (accommodated for distance)	10
„ posterior surface of lens (accommodated for distance)	6
Distance from anterior surface of cornea :	
To anterior surface of lens	3.6
To posterior surface of lens	7.5
To principal focal point	22.2

The nodal points are only 0.4 mm. apart, and may be replaced by an optical centre situated at the posterior surface of the lens.

The part of the retina which is most sensitive is that known as the 'yellow spot,' and for accurate vision it is necessary that the retinal image should be formed on this.

The angle alpha and the angle gamma (fig. 115).—An imaginary line drawn through the centre of the cornea, through the nodal point to the posterior pole of the eye, is called the *optic axis*; it divides the eyeball into symmetrical halves, and is, therefore, the geometrical axis of the eye (fig. 115, $A A'$).

The *visual axis* ($O F$) is the line of connection between the object looked at and the yellow spot; it passes through the nodal point n , crossing the optic axis, and emerges from the eye internal to the centre of the cornea. A line drawn from the object looked at to the centre of rotation is called the *line of fixation* ($O r$). The angle formed by the line of fixation with the optic axis is called the *angle gamma* (γ); the size of this angle varies with the refraction of the eye: it increases in hypermetropia; it diminishes in myopia, so that sometimes the line of fixation coincides with the optic axis, or even a negative angle may be formed by the line of fixation being external to the optic axis. These alterations in the angle gamma give rise to apparent squints (see Strabismus), convergent in myopia, divergent in hypermetropia.

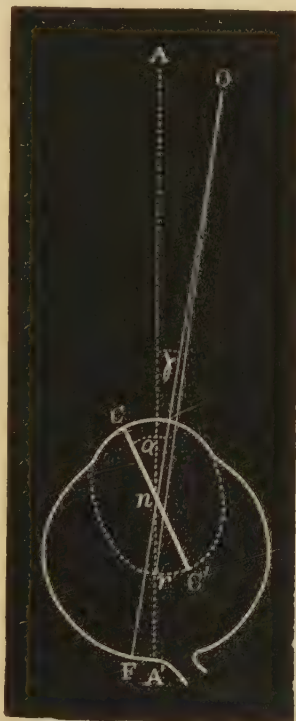


FIG. 115.—The Angles 'Alpha' and 'Gamma.'

The centre of rotation does not always lie on the optic axis, and it is more correct to define the angle gamma as formed by the line of fixation and a line joining the corneal centre and the centre of rotation. Landolt has given the name of *kappa* to the angle formed by the visual axis and the pupillary or optic axis, and it is to alterations in this angle that apparent squints are due.

The cornea forms part not of a sphere, but of an ellipse, the major

axis ($c c'$) of which meets the cornea, as a rule, just outside the optic axis. The angle formed by the major axis of the corneal ellipse with the visual axis is called the *angle alpha* (a). The knowledge of the latter angle is of value in explaining the cause of apparent deviations when the angle γ is found upon examination to be its natural size. The size of the angle γ is estimated by the use of the perimeter; it is in emmetropia about 5° .

In distant vision the visual axes are parallel, the optic axes diverge, and so, to a still greater extent, will the major axes of the corneal ellipses; the light falling on their summits gives to the eyes an appearance of divergence. Similarly, by a reversion of these corneal points an appearance of convergence may be noticed.

The *dioptric system* of the eye consists, then, of three refracting surfaces—the cornea, and the anterior and the

posterior surfaces of the lens; and of three refracting media—the aqueous, lens, and vitreous; the first and third, however, have the same index of refraction. Rays entering the eye are refracted at each of the three refracting surfaces, each refraction rendering them more convergent. In front of the lens is the iris, which forms a diaphragm whose aperture can be varied.



FIG. 116.—Frost's Artificial Eye.

Artificial eye.—Adams Frost has invented a very useful instrument in the form of an artificial eye for learning the practical use of the ophthalmoscope and for demonstrating the optical properties of the natural eye (see fig. 116).

Its simplicity makes it useful to all, even to those who do not possess more than the most elementary knowledge of optic principles. It is an inexpensive instrument, and therefore easily obtainable.

The method of estimating and correcting errors of refraction can

be completely mastered by its use. It is, besides, of great value in teaching students the use of the ophthalmoscope when it is impossible to obtain patients.

The dioptric system of the eye is represented by a biconvex lens of 40 mm. focus. Immediately in front of this is a disc containing diaphragms of several sizes, to represent different-sized pupils. By means of a rack and pinion the length of the eye can be varied between 30 and 65 mm., the distance of the retina from the posterior nodal point being shown by an index on a scale. There are two surfaces to represent the retina: the one, a ground glass, divided into millimetres—so that the formation of images can be seen and their size measured; the other painted to represent a normal fundus. In front of the lens are two fixed clips A and B, placed respectively at 5 and 10 mm. from the anterior nodal point; while a third (C) clip travels on a graduated bar which can itself be lengthened, shortened, or removed, and is constructed to hold a lens, test-object, or a ground-glass screen.

Tables showing mode of representing various degrees of ametropia are upon the foot-support of the artificial eye, although they are not shown in the figure.

MYOPIA		HYPERMETROPIA	
Correcting Lens in A	Length of Eye	Correcting Lens in A	Length of Eye
-1 D	41·66	1 D	38·45
-2 D	43·47	2 D	37·0
-3 D	45·36	3 D	35·65
-4 D	47·44	4 D	34·38
-5 D	49·7	5 D	33·19
-6 D	52·17	6 D	32·06
-7 D	54·83	7 D	31·0
-8 D	57·77	8 D	30

To represent Emmetropia, adjust the artificial eye so that the index points to 40—the retina is at the principal focus of the lens, when the eye will be emmetropic. If the ground-glass retina is placed at the back, and the eye directed to some distant object, it will be clearly seen as an inverted image on the glass retina. This is the appearance seen on the ground-glass or focussing screen of a camera while taking a landscape photograph. Direct the eye to some near object; the retinal image is out of focus; if a convex lens be now placed in A, its focus coinciding with the position of the object, the latter will form a clear image on the retina. In the living eye this *accommodation* for near objects is accomplished by

increasing the convexity of the crystalline lens. In this respect the eye differs from a photographic camera, which otherwise it closely resembles. In taking a portrait, the camera is accommodated for near objects by increasing the distance between the optical centre of the lens and the focussing screen. The artificial eye with a glass retina is practically a diminutive camera; the natural eye is analogous, the retina taking the part of the sensitive plate; the chemical change, however, produced in the retina by the images of external objects is transitory, whereas in a sensitive plate it is permanent.

Remove the ground-glass retina and affix the painted retina. In looking directly into the eye without an ophthalmoscope, nothing can be seen, all is dark within, because the observer's head intercepts the rays of light which should enter the eye, so that by their reflection from the retina the details of the fundus may be seen. The ophthalmoscope was invented to overcome this difficulty: the mirror reflects the light into the eye and the central aperture in it allows the returning rays to enter the observer's eye.

On ophthalmoscopic examination by the direct method, the details of the fundus (painted retina) can be clearly seen if the observer can completely suspend his accommodation, otherwise the details will be ill-defined and only clearly seen by adding concave lenses. It is very difficult at first to relax one's accommodation upon looking at so near an object. The image of the fundus is erect and magnified. If the head be gradually withdrawn from the eye the details will disappear, though the whole pupillary area maintains its illumination; owing to the rapid divergence of the issuing axial rays, and consequently the rays parallel to them, only a small area of the fundus is seen, yet sufficiently magnified to illuminate the whole pupil.

By the indirect method the fundus details are seen inverted. This is due to the fact that the eye has been rendered so myopic by the strong convex lens held in front of it that its far-point is at the principal focus of the lens (about 3 inches); this image is seen by the observer.

Replace the ground-glass retina, and fix in the rod supporting the clip c. Place in A the lens + 10 D. If the ground-glass screen be placed in c 10.5 cm. from the eye, and the latter placed with its retina towards a good light, an inverted image of the markings on the retina will be formed at its conjugate focus on the ground-glass screen.

Myopia can be produced by lengthening the artificial eye to more than 40 mm. Since the retina now lies beyond the principal focus of the lens, only diverging rays can be focussed on the glass retina. Retain the ground-glass screen in the clip c, but remove the strong

convex lens. Move the screen along the bar until the cross lines on the glass retina become clearly visible and distinct upon it; this is the punctum remotum (*p.r.*, or far-point) of the eye. The longer the eye is made, the shorter the distance of its *p.r.* Replacing the painted fundus and examining by the direct method, no definite details will be seen unless concave lenses be used; the lowest concave lens with which the fundus details are distinct is the measure of the amount of myopia. If, using the mirror alone, the head be withdrawn, the details of the fundus will be seen distinctly as an inverted image; this is the real, inverted, aerial image corresponding to the *p.r.* of the eye. The 'far-point' of the myopic eye can be found by the following formula, *F* being the principal focal distance of the lens = 40 mm., *f* the distance of the retina from the lens, *p.r.* the 'far-point'; then

$$\frac{1}{p.r.} = \frac{1}{F} - \frac{1}{f}.$$

So that, if the eye were lengthened to 50 mm., the *p.r.* would be at 200 mm.—

$$\frac{1}{p.r.} = \frac{1}{40} - \frac{1}{50}; \text{ i.e. } p.r. = 200 \text{ mm.}$$

In order that a lens shall correct the myopia, it is necessary for its principal focus to coincide with the 'far-point,' so that it will render parallel rays divergent as if they came from that point.

In indirect ophthalmoscopic examination the image will increase in size as the lens is withdrawn from the eye.

By retinoscopy, the shadow will be seen to move in the same direction as the rotation of a concave mirror. Replace the glass retina and fix the ground-glass screen at the *p.r.* of the myopic eye. Impinge upon the back of the glass retina by means of a convex lens a circular diffusion image of some luminous source; an image of it is formed on the glass screen. Make the diffusion image on the glass retina move upwards; the image on the screen will move downwards; *i.e.* the image at the punctum remotum moves in an opposite direction to the movement of the diffusion image on the retina.

To represent Hypermetropia, shorten the eye to less than 40 mm.; the ground-glass retina now lies within the principal focus of the lens; only convergent rays, then, can come to a focus on the retina, and since no such rays exist in nature it is impossible to see any clear images of distant objects on the glass retina.

By direct ophthalmoscopic examination the details of the fundus (painted retina) can be distinctly seen without the use of any lens in the ophthalmoscope, because the observer can, by accommodating,

focus diverging rays on his retina—*i.e.* by increasing the strength of his own lens and so making up for the deficiency which exists in the artificial eye or the eye of any person under examination. The accommodation will give way *pari passu* with the addition of convex lenses, allowing the fundus details to be seen distinctly with several different lenses; the highest, however, with which the details are seen clearly is the measure of the amount of hypermetropia.

If, using the mirror alone, the observer withdraws his head, he will see an erect image of a considerable portion of the fundus, owing to the fact that the emergent rays of a hypermetropic eye are divergent, and that diverging rays from any one point on the retina intermingle with similar diverging rays of adjacent pencils of rays; consequently, a virtual image of the fundus can be seen.

If F is the principal focal distance = 40 mm., f the distance of the retina from the lens, and *p.r.* the 'far-point,' the latter may be found by the following formula:

$$\frac{1}{p.r.} = \frac{1}{f} - \frac{1}{F}.$$

The shadow, by retinoscopy, will be seen to move in an opposite direction to the rotation of a concave mirror. For an explanation of this phenomenon see Section VI.—Shadow Test.

Astigmatism may be produced by adding a cylindrical lens. Lengthen the eye so that the index again points to 40 mm., thus making it emmetropic. Place in front of it a convex cylinder lens, axis horizontal, so as to make the eye myopic in the vertical meridian. It is now astigmatic, having different degrees of refraction in its two chief meridians. Affix the glass retina so that the cross lines correspond to the chief meridians. If an emmetrope now examines the eye by the direct method, he will notice that the vertical lines are distinct, whereas the horizontal lines which are parallel to the emmetropic meridian are out of focus. If concave lenses are used in the ophthalmoscope, the horizontal lines will come into focus and the vertical lines become indistinct. To understand this phenomenon see Section IV. Similarly, if the painted retina is examined, the vertical vessels in focus point to the condition of the refraction in the horizontal meridian, and the horizontal vessels in focus reveal the refractive condition of the vertical meridian. It is necessary to remember that the highest convex lens and the lowest concave are the estimates of the chief meridians in mixed astigmatism.

An experiment may be shown to demonstrate that the 'fundus-illumination' is oval in astigmatism.

The peculiar features of the different kinds of astigmatism may

be readily demonstrated upon the artificial eye by retinoscopy and ophthalmoscopy.

Optical defects of the normal eye.—The eye is sometimes spoken of as if it were a perfect optical instrument; this, however, is very far from being the case. It is, it is true, 'wonderfully well adapted to its purpose, for the range of its vision extends in a straight line from a few inches from the eye to an infinite distance, whilst with the eyes directed forward, objects can be seen, although indistinctly, which lie as much as 90° on both sides of the head, and this lateral range can be increased still farther on either side by a movement of the eyes—the head remaining fixed; and of course to a much greater extent by movement of the latter. The perfect adaptation of the eye to all the requirements of vision does not depend so much on its perfection as an optical instrument as on its free mobility, the great sensibility of the retina, and the readiness with which the mind interprets the impressions conveyed to it.

Spherical aberration is to a great extent, but not entirely, obviated by the iris, and chromatic aberration is considerable. The cornea is not a perfectly spherical surface,¹ for its vertical meridian generally has a shorter radius of curvature than the horizontal. In consequence of this few people see vertical and horizontal lines with quite equal clearness. There is also a slight amount of irregular astigmatism present in the normal eye, which gives the radiate appearance to stars. The media of the eye, moreover, are not perfectly clear, for in the lens are numerous striæ and spots, besides the regularly radiating striæ which mark its division into sectors, and in the vitreous are a large number of floating cells and fibres. All these structures can be brought into view by throwing light into the eye in an unusual manner so that their shadows are formed on a part of the retina unaccustomed to them. The retina, too, does not form a continuous surface for the reception of visual impression, for besides the large hiatus formed by the entrance of the optic nerve—the 'blind spot'—the blood-vessels of the retina itself pass in front of its sensitive elements and cause linear gaps in the visual field.

The sensibility of the retina varies very greatly at different parts. So great is it at the centre—the fovea centralis—that the average eye can distinguish two points if they are separated by an angular measurement of only one minute, while many eyes can do the same with a somewhat smaller angle; but from this point towards the

¹ Strictly speaking, the cornea is not a portion of a spherical surface at all, but forms the extremity of an ellipse. A small circle described on the blunter extremity of an egg would give a good idea of its form.

periphery its sensibility rapidly diminishes, owing to the greater scarcity of the cones, so that as an object is moved towards the peripheral part of the visual field, its colour is first lost and then its form, although a visual impression, sufficient to indicate the presence of the object and its position, remains longer.

The eye can be so readily directed towards an object, so that its image falls on the fovea centralis, that this indistinct lateral vision is all that is really required for practical purposes; while, owing to our visual impressions being formed as the result of experience, we notice the defects in the visual field so little that most people are quite unaware that they have in each field a gap sufficient to include a man's head at a distance of seven feet.

Visual Angle.—It will be seen from what has preceded that the distance between the retinal images of any two points will



FIG. 117.—Visual Angle.

depend not only on the distance of the two points from each other, but also on their distance from the eye.

Thus in fig. 117, the retinal image $b'a'$ would occupy the same position at whatever points on the line aa' and bb' the points a and b were situated; and if a and b were the terminal points of an object ab , the retinal image of the object would be of the same size as long as it subtended the angle akb .

The angle made by the axial rays from the terminal points of an object at the optical centre is called the *visual angle*. The size of the retinal image of an object is in direct proportion to the visual angle under which it is seen; therefore, objects which are seen under the same visual angle have retinal images of the same size.

Visual Acuteness.—It is essential to have a standard of normal vision, and some method of expressing numerically departures from it. This is very conveniently supplied by Snellen's

test-types, which are those in ordinary use in this country, and of which a copy will be found at the end of this book. These consist of letters of various sizes, the strokes of which the letters are formed being in every case a fifth of the diameter of the letter. The smallest letters are 9 mm. in diameter, and at 6 metres are therefore seen under a visual angle of five minutes ($5'$); while each component stroke is seen under an angle of one minute (see fig. 118). This has been found to be the smallest visual angle under which the majority of healthy eyes can recognise an object. If, therefore, the row of smallest letters can be distinctly seen at 6 M, the visual acuteness is said to be normal, or it may be expressed as $V = 1$.



FIG. 118.
No. 6 Snellen.

Each row of letters has a number indicating the distance at which it must be placed, in order to be seen under a visual angle of $5'$, and the visual acuteness may be conveniently expressed by a fraction, the numerator of which is the distance in metres at which the letters are situated, and the denominator the distance at which the smallest letters which can be read would make a visual angle of $5'$.

For example: (1) Standing at 6 M, the smallest letters are read $V = \frac{6}{6}$. (2) But, if at 6 M the smallest letters which can be read are those which would make a visual angle of $5'$ if removed to 12 M, it is evident that the visual acuteness is only half that of the normal eye, $V = \frac{6}{12}$. (3) The letters which should be seen at 60 M cannot be read until they are brought as near as 3 M, $V = \frac{3}{60}$, and so on.

In this country the distance is sometimes expressed in feet instead of in metres.

Besides the test-types just described, there are others which form a continuous series with them, and which are adapted for distances ranging from 5 M to 0.50 M. These, for distinction, are called Reading-Types, and a sample of them will also be found at the end of the book. They are not so well adapted for testing the visual acuteness as the distance-types, as for near objects the accommodation must be used, and a defect due to weakness of the latter might be mistaken for diminished acuteness of vision. Objects, on the other hand, held near the eye are rather more easily recognised than more distant ones

which are seen under the same visual angle, because the amount of light entering the eye in the former case is proportionately greater; for, while the size of the retinal image varies directly as the distance, the amount of illumination varies as the square of the distance. Notwithstanding this source of error, the reading-types often form a very convenient rough test of the visual acuteness. In this country Snellen's reading-types are less used than those of Jaeger, which are not arranged on any scientific plan, but are simply ordinary printer's types of various sizes from 'Brilliant' to '8-line Roman,' numbered consecutively.

Section III.—NORMAL REFRACTION, OR EMMETROPIA.

DEF.—*An emmetropic eye is such that all parallel rays entering it while at rest are so refracted that they come to a focus on the retina (fig. 119).*

Thus, the retina of an emmetrope is situated at the principal focus of the dioptric system.



FIG. 119.—Rays entering an Emmetropic Eye.

This refractive condition of the eye is taken as the normal, since it possesses the greatest range of visual power.

An eye at rest is one in which all accommodation is suspended, and is the lowest refraction of any given eye. Rays from near objects in an emmetropic eye at rest do not come to a focus on the retina, but (if produced) beyond it (fig. 119, dotted line).

Since rays from a distance of six metres or beyond enter any eye, for all practical purposes, parallel, objects at or beyond that distance, which subtend an angle not less than 5', can be distinctly recognised as to their form and nature by an

emmetropic eye. The far-point (often written *p.r.*, *punctum remotum*), therefore, of an emmetrope may be considered as at infinity.

The near-point (*punctum proximum*, *p.p.*) varies with age. (See Accommodation.)

If a biconvex spherical lens be placed in front of an emmetropic eye (fig. 120), parallel rays will be rendered convergent



FIG. 120.—Rays entering an Emmetropic Eye through a Convex Lens.

upon entering it, and will come to a focus in front of the retina. Distant vision will be subnormal. It is evident that accommodation would make matters worse.

Rays, however, coming from the principal focus of the lens, or from any adjacent point in the same vertical plane, will come

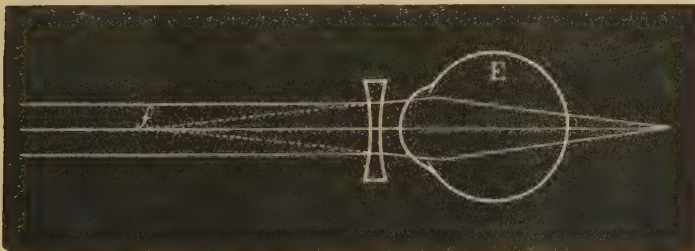


FIG. 121.—Rays entering an Emmetropic Eye through a Concave Lens.

to a focus on the retina (see fig. 120, dotted line) ; *f* is the real principal focus of the lens.

Conversely, if a biconcave spherical lens (fig. 121) be placed in front of an emmetropic eye, parallel rays will be rendered divergent upon entering it, and will not come to a focus on the retina, but, if prolonged, behind it. Distant vision will be

subnormal. If, however, accommodation be brought into play, it will rectify the visual defect so long as the lens does not exceed a certain strength. In fig. 121, f is the virtual principal focus of the lens.

From the foregoing we may conclude that an emmetropic eye can see $\frac{6}{6}$; a low convex sphere will lower its visual power, and a low concave will *not* do so unless the accommodation be suspended, as when an eye is atropinised, or by old age. An emmetrope, if atropinised, still maintains perfect distant vision.

It is easy to understand that parallel rays may come to a focus before they reach the retina, or come to a focus only if produced behind the retina.

Thus, in hypermetropia the retina is in front of the principal focus of the dioptric system (fig. 122, H), whereas in myopia it is beyond it (M).



FIG. 122.—Position of the Retina in Emmetropia and Ametropia.

Emmetropia, therefore, forms the unit or mean refraction (fig. 122, E), while any departure from this is called an *error of refraction*, or *ametropia*.

Accommodation.—So far the eye has been described as a passive instrument in which images of distant objects only can be formed with any clearness on the retina, for the latter is placed at the principal focus of its dioptric system. The eye, however, possesses the means of increasing its refractive power, and so adapting itself for near objects, but there is considerable difference of opinion as to the exact mechanism of accommodation. All are, however, agreed that during the act of accommodation for near objects, the anterior surface of the lens becomes more convex. The capsule of the lens is attached by the suspensory ligament to the ciliary processes, and any alteration in its tautness will produce an alteration in the form of

the elastic lens. According to the, until lately, widely held theory of *Helmholtz*, during the act of accommodation for near objects, the suspensory ligament is relaxed. This is chiefly brought about by the annular part of the ciliary muscle, which on contracting causes the ciliary processes to approximate towards the axis of the eye. Help is also given by the meridional fibres which, acting from the sclero-corneal junction as a fixed point, draw the choroid and ciliary body forwards. As a result, the ligament is relaxed, and the elasticity of the lens substance causes the whole of its anterior surface to become more spherical in shape. Recently, however, *Tscherning* has asserted that the suspensory ligament is tightened, not relaxed, during accommodation. He considers that the meridional part of the ciliary muscle uses the choroid as its fixed point, and draws the ciliary processes towards this. The periphery of the lens is consequently flattened by the taut ligament, the centre of the lens surface becoming more convex, the shape of the whole anterior surface becoming hyperboloid. *Schön* agrees with *Tscherning* in the main, but gives the most important rôle to the annular fibres, which he believes act as a ring of pressure round the equator of the lens during accommodation. The anterior pole protrudes forwards, but increased vitreous pressure prevents any alteration in the shape of the posterior pole. At the present time, the evidence is rather in favour of the older theory of *Helmholtz*.

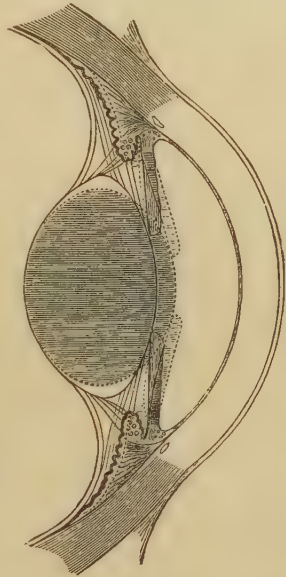


FIG. 123.—Diagram illustrating *Helmholtz's* Theory of Accommodation. (After *Landolt*.)

The muscular act of accommodation has always associated with it a contraction of the pupil, which prevents the most divergent rays from entering the eye.

Amplitude of accommodation (A.A.) is the total accommodative power of any given eye. It must be distinguished from the *range of accommodation*, which is the distance between the punctum remotum (*p.r.*) and punctum proximum (*p.p.*) They both vary physiologically with age.

As age advances, the substance of the lens becomes less elastic, and the same muscular effort does not then produce so great an increase in its convexity. At the age of ten the accommodation is sufficiently powerful for an object to be clearly seen at 7 cm. ($2\frac{3}{4}$ in.), but after this it gets gradually weaker, so that the nearest point of distinct vision (often written *p.p.*, punctum proximum) recedes farther and farther from the eye, until, at the age of seventy-five, all accommodation is lost.

A knowledge of the strength of accommodation proper to each period of life is necessary in order that any departure from the normal condition of this function may be recognised.

In the following table, opposite each age, is placed in the first column the strength of the lens, in dioptries, which is equivalent to the maximum amount of accommodation which can be used, and which therefore expresses the *amplitude of accommodation*. In the last two columns are given the distances of the 'near-point' in centimetres and inches.

Table of Amplitude of Accommodation. (From Landolt.)

Age	Amplitude of accommodation	Distance of 'near-point'	
		cm.	ins.
10	D 14	7	$2\frac{3}{4}$
15	12	8	3
20	10	10	4
25	8.5	11.7	$4\frac{1}{2}$
30	7	14	$5\frac{1}{2}$
35	5.5	18	7
40	4.5	22	$8\frac{3}{4}$
45	3.5	28.6	$11\frac{1}{2}$
50	2.5	40.5	16
55	1.75	57	23
60	1	100	$39\frac{1}{2}$
65	0.75	—	—
70	0.25	—	—
75	0.0	—	—

The amplitude of accommodation (A.A.) is measured as follows: Let it be supposed that the person is emmetropic, and

that his *p.p.* is 10 cm., then the unit of accommodative power, one dioptré—equivalent to one metre—divided by his *p.p.* in centimetres, is the method for finding his A.A.; thus:

$$\frac{1 \text{ D}}{10 \text{ cm.}} \text{ or } \frac{100}{10} = 10 \text{ D of A.A.}$$

Since his *p.p.* is situated at 10 cm., the rays from that point enter his eye divergent; the lens which renders these rays parallel upon entering the eye is a convex sphere with its principal focus situated at his *p.p.*—*i.e.* + 10 D (see p. 481).

Therefore, *the highest convex lens with which a person can see clearly small reading-type at the distance of his punctum proximum is the measure of his amplitude of accommodation.*

The A.A. may also be estimated by concave lenses; the strongest concave lens with which an emmetrope can see $\frac{6}{8}$ is the measure of his amplitude of accommodation.

A hypermetrope uses some of his accommodation so that he may see $\frac{6}{8}$; the amount he uses (the total error) must be added to the A.A. found, according to the method adopted in an emmetrope. Thus: H=5 D, *p.p.*=20 cm.,

$$\frac{100}{20} + 5 \text{ D} = 10 \text{ D} = \text{A.A.}$$

If A.A. is known, and also the amount of H, the *p.p.* can be found.

Let H=6 D, A.A.=10 D.

Since 6 D of H is used up for distant vision,

$$10 - 6 = 4 \text{ D only for reading, } \frac{100}{4} = 25 \text{ cm.} = \textit{p.p.}$$

In myopia, the amount of error must be subtracted from the A.A., as estimated by the position of the *p.p.*

Perhaps a better and easier plan is first to correct the error, and then to estimate the A.A. by the new position of the *p.p.*

Fig. 124 illustrates the differences between the amplitude and the range of accommodation under different refractive conditions and at different ages. The line *n* passes through the nodal point of each eye. The vertical lines indicate, in centimetres, different distances from the nodal point. The

thick horizontal lines show the range of accommodation in dioptries, from which it will be seen that in A, an emmetropic eye, and D, a very myopic eye, both belonging to persons of the same age and the same amplitude of accommodation (10 D), a very marked difference exists in the *range* of accommodation, the latter possessing only 14.4 cm. B is an emmetropic eye of a person aged fifty; the range is slightly less than in A, but the A.A. is decidedly less, being only 2.5 D. C is a myopic eye of a person aged about thirty-seven, who possesses only half as much A.A. as D, but over twice the range.

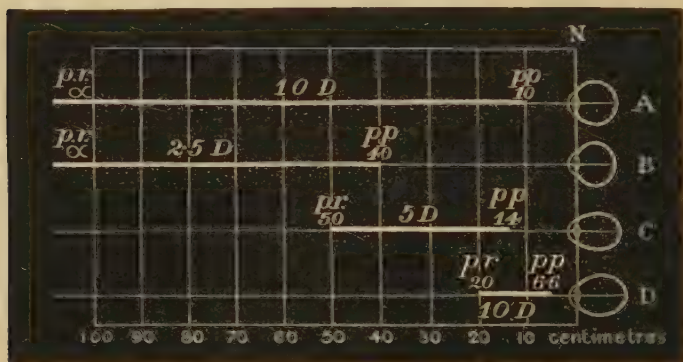


FIG. 124.—Differences between the Amplitude and the Range of Accommodation.

When the near-point recedes farther than 25 cm. (10 in.) reading, &c., becomes difficult, because, at the distance at which the book is ordinarily held, the whole accommodation available has to be used, and hence fatigue is soon experienced—*accommodative asthenopia*; while if the book is held farther away, only large print can be read, because of the diminution in the size of the retinal images.

When, owing to the failure of accommodation, the 'near-point' has receded beyond 22 cm. (9 in.), the condition is called *presbyopia* (aged sight), and its effects are obviated by supplementing the accommodation by convex spectacles of such a strength as to bring the near-point back to 22 cm. If the refraction of the eye is normal, the lens which will be required to do this will depend on the age of the patient. The presby-

opic correction proper to any age can be found by ascertaining the difference between the amplitude of accommodation which corresponds to that age and 4.5 D, which is the amount required to bring the near-point to 22 cm. A useful practical rule to remember is to add 1 D for every five years after forty; thus an emmetrope at forty-five would require sph. + 1 D; at fifty, sph. + 2 D; at fifty-five, sph. + 3 D; at sixty, sph. + 4 D, for reading and near work only; beyond the age of sixty, how-

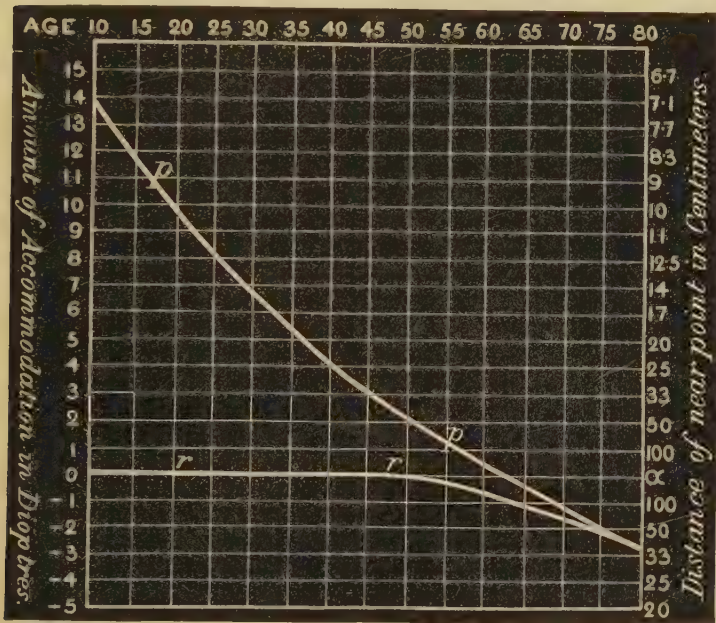


FIG. 125.—Diagram showing the Range of Accommodation at different Ages. (After Donders.)

ever, this does not hold good. If the refraction is not normal, it must be corrected first, and then the presbyopic correction added to the glass which is required for this purpose. In cases where the patient requires to see at his work at a greater distance than that at which a book is generally held, glasses slightly weaker than what would correct the presbyopia must be given; in such cases, however, the patient is generally the best judge. This subject will be referred to again in a later section.

Loss of accommodation may occur as a pathological affection from defective action of the ciliary muscle, as cycloplegia (see Chap. XV.), spasm, or hysteria; or from absence of the lens, as after solution or extraction for cataract, or when the lens is dislocated.

Convergence is the symmetrical rotation inwards of the two eyes, so that the visual axes may meet at any point desired

between infinity and a few centimetres from the nose. It may be defined as the *maintenance of binocular vision for near objects*.

The *range of convergence* is the difference between the position of the visual axes for the 'near' and 'far points' of binocular vision. It is measured by 'metre-angles.' In fig. 126, let L and R be the left and right eyes of any person, with their visual axes parallel, directed to a and a' . Let the eyes converge to an object A at one metre distance: this is the unit of convergence, and is called *one metre-angle* ($1\ ma$); at B, half a metre distance, the angle LBR is twice the size of the angle LAR; it is therefore called *two metre-angles* of convergence, *i.e.* the measure of the metre-angle is the inverse ratio of the distance; just as the measure of

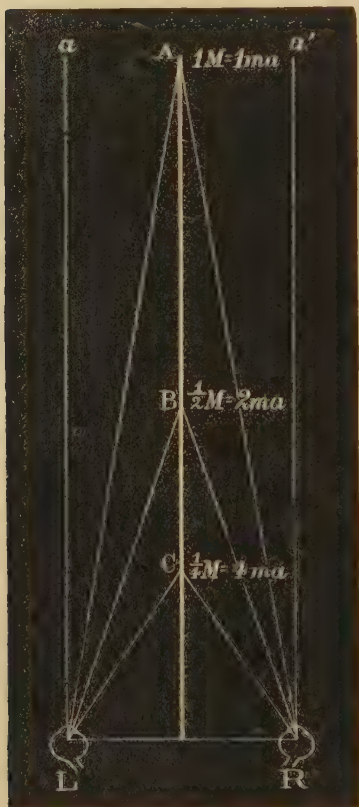


FIG. 126.—The 'Metre-angle.'

the amplitude of accommodation is, in dioptries, the inverse ratio of the distance.

The *absolute* value of the 'metre-angle' depends on the distance between the two eyes, and is constant for each individual. In a person whose eyes are 7 cm. apart, the metre-

angle has a value of 2° . If the eyes are 6.4 cm. apart, the metre-angle is equivalent to $1^\circ 50'$.

Since accommodation and convergence are associated acts under the control of a common centre in the brain, it is necessary to make a few remarks about their relationship to one another. This is not absolute; there is a slight margin left, so that a little more or less convergence may occur with any given distance of accommodation. This margin is greatly augmented in refractive errors, whilst in emmetropia it is slight.

Thus, an emmetrope wishing to see an object at a distance of 20 cm. would have to converge $\frac{100}{20} = 5$ *ma*, and to accommodate 5 D.

In hypermetropia accommodation is necessary in order to see distant objects; if $H = 2$ D, 2 D of accommodation is required for distant objects; therefore 2 *ma* of convergence ought to be brought into play. This would produce diplopia, since the images on the fundus would be formed on non-corresponding areas of the retina; so, to avoid a convergent strabismus, convergence must be kept in abeyance for distant objects. In looking at near objects there must, of necessity, be a similar altered relationship between these two forces.

In myopia, on the other hand, no accommodation is required for objects placed at the *p.r.*, which, being at a limited distance, necessitates convergence in order to allow the visual axes to meet at that point; if $M = 1$ D, convergence = 1 *ma*. Accommodation ought to be 1 D, but it is not needed. If accommodation could not be kept in abeyance without suspending convergence, it is easy to conceive that divergence might ultimately ensue.

The *maximum and minimum of convergence* are the extremes of the range of convergence; the former has an average of 9.5 *ma*, the latter is a negative quantity equivalent to -1 *ma*.

The measurement is made by prisms, and in order to appreciate their value it will be necessary to give a brief description of them.

A *prism* is a wedge of transparent substance, possessing

at least two plane surfaces, which are divergent and enclose an angle. The thick end of a prism is called its base, the thin end its apex.

Rays of light passing through a prism are refracted towards its base (see fig. 106, Sect. I.).

The amount of refraction depends upon the strength of the prism. The strength is equal to half the number of any prism. Prisms are numbered according to the size of the angle formed

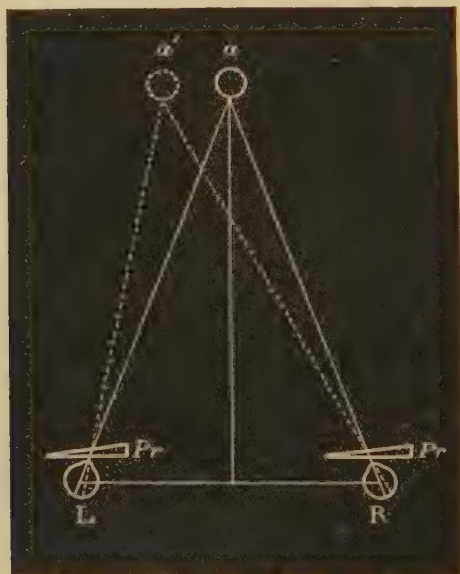


FIG. 127.—Diplopia produced by Prisms.

by the diverging surfaces; thus, a prism of 10° has a deviating strength of 5° . This method of numbering prisms is inaccurate and unscientific, since the material of which the prism is made is neglected, although the deviation produced varies with the refractive index of the glass. Landolt numbers prisms according to the deviation produced, and consequently a prism marked ' 10° ' has a deviating strength of 10° , and not about 5° according to the old nomenclature. Objects when looked at through a prism appear to be displaced in the direction of its apex.

Example.—Hold a prism of 12° in front of the right eye, with its *base inwards*, and look at a distant object; homonymous diplopia is produced, *i.e.* the image is to the right of its object, displaced to the same side as the eye under cover of the prism. Now converge, and the image and object will separate. Reverse the prism, *base outwards*—crossed diplopia occurs. Now converge, and the image and object will fuse, *i.e.* a prism of 12° base outwards is easily overcome by convergence.

If in fig. 127 a prism (*Pr*) be placed in front of the right eye (*R*) with its *base out*, the object *a* will form a retinal image on the fundus of *R*, external to the yellow spot, and so an image

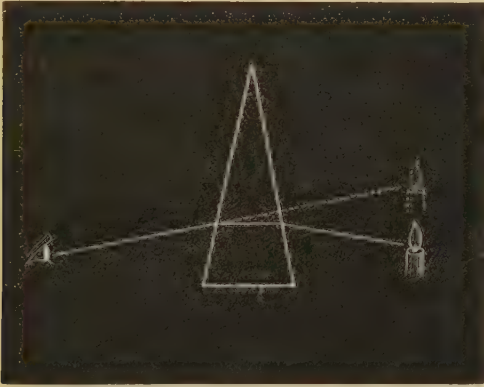


FIG. 128.—Virtual Image caused by a Prism.

a' will be perceived to the left of its object in the visual field (crossed diplopia). Similarly, if a prism be placed in front of *L* with its *base in*, homonymous diplopia is the result, because *L* perceives *a* as if it came from *a'*, *i.e.* the object appears to be displaced in the direction indicated by the apex of the prism.

If two prisms of the same strength be placed before the eyes, one *base in* and the other *base out*, as in fig. 127, lateral displacement of the image occurs, but no diplopia.

Though the object is received on the yellow spot of the observer, yet an apparent displacement of it towards the apex of the prism is noticed. This is the virtual image of the object (see fig. 128). If a prism, base in, be placed in front of one eye of a person who is fixing some distant object, in order to

avoid diplopia he turns the eye out so as to allow the object to fall on the yellow spot. The strongest prism, *base in*, that does not produce diplopia of a distant object is the measure of the minimum of convergence; at the same time, it is the measure of the maximum divergence. (This, in emmetropia, is usually equivalent to a prism of 8° , or a prism of 4° in front of each eye.) Since the eyes are divergent, the rays prolonged backwards would meet behind the eyes at a distance of about 1 metre; the minimum convergence can thus be acknowledged as -1 ma .

Conversely, the highest prism, base outwards, which does not produce diplopia upon looking at an object as near as accommodation will permit, is the estimate of the maximum convergence. It is equal to about 9.5 ma .

It is evident from what has been said that prisms with their *bases inwards* relieve convergence, and with their *bases outwards* promote convergence. Consequently, prisms are occasionally prescribed in practice. (See Heterophoria.)

A ready test for finding the maximum convergence is to see how near to the nose

the patient can fix with both eyes a vertical black line on a sheet of white paper, without diplopia. A more accurate estimation, and one more simple than the prism test, is based upon this principle. The measurement is made by the use of Landolt's dynamometer (fig. 129), which, described in his own words, 'consists of a cylinder, blackened on the outside, which can be fitted on a candle of ordinary size. The cylinder has a vertical slit about a third of a millimetre in breadth, a series of fine openings which form together a vertical line, and a circular aperture about one millimetre in diameter. The slit and the openings are all covered with ground glass. When

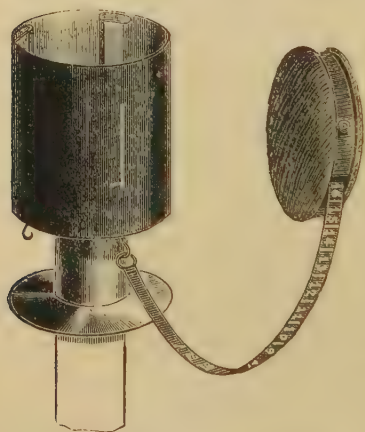


FIG. 129.—Landolt's Dynamometer.

the candle is lighted, they constitute luminous objects of fixation. Beneath each opening can be attached one end of a tape measure that is rolled up by means of a spring. The tape is graduated on one side in centimetres, on the other in the corresponding numbers of metre-angles (or, what amounts to the same thing, in dioptries).

‘To determine the *maximum of convergence*, we use the luminous slit as the object of fixation. The tape measure is drawn out to about seventy centimetres, its case being held beside one of the eyes of the patient (theoretically, on a level with its centre of rotation), while the object of fixation is placed in the *median line*. If the patient sees the object single, then, by pressing on the knob of the case, the spring is made to roll up the tape, and thus the observer brings the fixation-object nearer to the eyes, taking care, however, that it always remains in the median line. So soon as the person under observation begins to see double, the *near point of convergence* is attained. In fact, when the eyes have no longer the power required to fix the object simultaneously, there exists a divergence relatively to its position, and, consequently, crossed diplopia.

‘At this instant, one side of the tape gives in *centimetres* the distance of the *punctum proximum* of convergence, and the other side the corresponding *maximum of convergence* in *metre-angles*.’¹

Decentration of spherical lenses.—A section of a spherical lens presents the appearance of two prisms, either with their bases touching (convex) or their apices touching (concave). If a sphere be displaced inwards or outwards, so that the visual axis does not pass through its centre, it is clear that the lens will possess a prismatic action upon rays of light entering the eye. If a prism is required for any person wearing spherical lenses, it is only necessary to *decentre* the sphere to produce the combined effect. Convex lenses displaced inwards, and concave outwards, act as prisms with their bases in, and so relieve convergence; convex lenses displaced outwards, and concave inwards, will act as prisms with their bases out, and

¹ Landolt, *Anomalies of the Motor Apparatus of the Eyes*; Oliver and Norris, *System of Diseases of the Eye*, vol. iv. p. 140 (1900).

so promote convergence. The amount of prismatic effect of a spherical lens depends upon its strength and displacement.

The value of prisms in paralytic affections will be described in the chapter on that subject.

Section IV.—ERRORS OF REFRACTION, OR AMETROPIA.

There are three forms of Ametropia—**Myopia**, **Hypermetropia**, and **Astigmatism**.

If parallel rays come to a focus before they reach the retina, the condition is called *myopia* (fig. 130).

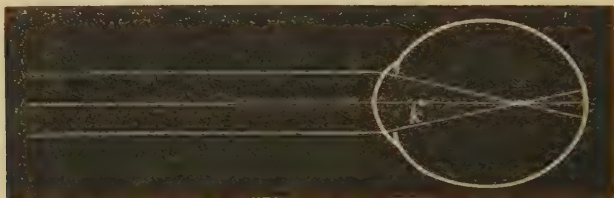


FIG. 130.—Parallel Rays entering a Myopic Eye.

If parallel rays do not come to a focus on the retina, but if prolonged behind it, the condition is called *hypermetropia* (fig. 133).

Thus, a myopic eye is apparently one with a higher refracting system, and a hypermetropic one with a lower refracting system.

If rays are refracted unequally in different meridians of the eye, the condition is called *astigmatism*.

Owing to occasional irregularity of the surface of the cornea from conicity or previous ulceration, rays may be irregularly refracted, and produce distorted images of external objects on the retina; this is known as *irregular astigmatism*, and is practically unassisted by glasses.

If rays are refracted equally in any one meridian, but unequally in different meridians, so that the meridians of the highest and lowest refractive powers are at right angles to one another, the condition is known as *regular astigmatism*, and is greatly benefited by the use of proper glasses.

It must be understood that in speaking of the refraction of

an eye the accommodation is always assumed to be relaxed, unless mentioned to the contrary.

(i) **Myopia** (fig. 130). DEF.—*Myopia is that condition in which parallel rays entering an eye at rest are so refracted that they come to a focus before they reach the retina—i.e. the retina lies beyond the principal focus of the dioptric system.* This may be due (1) to the antero-posterior axis of the eye being too long, or (2) to the refraction of the eye being too great. The first, which is called *axial myopia*, is by far the more common; the second, *refractive myopia*, may be due to (1) an increase in the refractive power of the nucleus of the crystalline lens, sometimes being met with in the early stages of senile cataract; (2) alteration in position of the



FIG. 131.—The Far-point of a Myopic Eye.

crystalline lens, being sometimes found in anterior displacement of the lens; and (3) conical cornea.

Since the retina lies beyond the principal focus of the dioptric system of the eye, rays from any point (*a*, fig. 131) on the retina do not leave the eye parallel, as in emmetropia, but converging (compare fig. 111, on p. 461), and they will therefore meet at a focus (*a'*) in front of the eye. Conversely, the only rays which can come to a focus on the retina, while the accommodation is at rest, are diverging rays from points which lie in the same plane as *a'*, as, for instance, *b'*. Since rays coming from *a'* and *b'* come to a focus at *a* and *b* respectively, it follows that a retinal image would be formed of any object of which *a* and *b* were the terminal points.

If the accommodation were used, rays which were more divergent—i.e. coming from a nearer point than *a'*—could be brought to a focus on the retina; but under no circumstances could this occur with those which are less divergent—i.e. coming

from a greater distance than a' . For this reason a' is called the '*far-point*' of the eye, as it is the farthest point of distinct vision, and may be defined as the conjugate focus of the yellow spot; it is positive and finite.

Not only is an image of an object, which is situated at the far-point of a myopic eye, formed on the retina, but a real inverted image of the fundus is formed at the far-point (see p. 457).

We have seen that to the emmetrope distance alone forms no limit to vision; the myope, on the contrary, has clear vision of no objects situated beyond his far-point: hence the popular name for myopia—'*short-sightedness*'—is a good one.

Rays, coming from a point beyond the far-point of a myopic eye, can be focussed on the retina by rendering them as



FIG. 132.—Parallel Rays entering a Myopic Eye through a Concave Lens.

divergent as they would be if they came from the '*far-point*;' for parallel rays this would be accomplished by a concave lens of such a strength, and placed in such a position, that its principal focus would coincide with the '*far-point*.' Thus, in fig. 132, the far-point is at pr ; rays, therefore, which diverged from this point would be focussed on the retina; if, now, a concave lens be placed in front of the eye, its focus being at pr , it will render parallel rays as divergent as if they came from that point, and so enable them to be focussed on the retina.

Myopia is, as we have seen, usually the result of an elongation of the antero-posterior diameter. The eye is generally also enlarged in other directions, but to a less extent. Although the tendency to myopia is frequently inherited, that condition is itself seldom present at birth, but comes on during childhood. The essential condition for the production of myopia would seem to be a diminished power of resistance in the ocular tunics; it

is, however, a disputed point what the anatomical lesion is which causes the weakening. Whether any such anatomical weakness, congenital or acquired, exists or not, it is certain that myopia is brought about by a pernicious habit of prolonged and excessive convergence, an abuse of one of the most important functions of the eye; and by the association which exists between it and accommodation, this latter and equally valuable function is similarly ill-used. The most potent cause which gives rise to this condition is the employment of the eyes in childhood for near work in a defective light. To compensate for the paucity of light an attempt is made to obtain larger retinal images. The head is held down close to the book; this necessitates a strong effort of convergence in order that binocular vision may be maintained, and a corresponding effort of accommodation is made by each eye. The action of the recti muscles on the globe tends, if its tissues are weak, to cause it to bulge at the posterior pole, where it is unsupported by muscles. The eye has not attained its full growth, and its circulation, like that of all growing organs, is active and easily influenced by causes which would not affect a fully developed organ. The obstruction to the return of blood by the compression of the cervical veins produced by the position of the head, and the action of the recti and ciliary muscles, induces a state of chronic congestion which weakens the investing tunics. In an exactly similar way, deficient vision due to opacities in the ocular media, such as corneal nebulæ and lamellar cataract, is to a certain extent remedied by the child by increasing the size of the retinal image by means of a strong effort of convergence and accommodation. There is possibly also combined with this, in some cases, an increased activity in the secretion of the intra-ocular fluid, so that, while on the one hand the power of resistance of the eye is diminished, on the other the forces tending to its distension are increased.

In the worst cases a chronic degenerative process is set up in the sclerotic and choroid at the posterior pole, and the elongation of the eye rapidly increases (*progressive myopia*, *sclerotico-choroiditis*, *myopic choroiditis*), while the choroid becomes thinned and atrophied, the changes usually commencing

in the portion adjacent to, and on the outer side of, the optic disc.

Clinically, myopia may be divided into *simple* and *progressive*. Simple myopia is by far the more frequent variety met with, and is characterised by the error being low in amount, by the absence of any marked fundus change, by the visual acuity being normal, and by having no tendency to increase after the proper correction has been prescribed. Simple myopia may increase very gradually, even after the glasses have been ordered; in such cases it will often be found upon inquiry that the glasses have been used only for distance and ignored for near work. Myopia is rarely seen before the age of ten; it occurs equally in the two sexes; is more common among Jewish than Christian races; is markedly hereditary, though very rarely congenital. Progressive myopia, often called malignant or pernicious myopia, is diagnosed by the necessity of frequent changes in the glasses worn, by the marked fundus changes, and by defective visual acuity. A constant aching pain may be present in both eyes, but is in no way characteristic. Prominence of the globes is sometimes seen, and the elongation of the eyeball may often be made evident by looking at its outer surface when the eye is turned in. Its onset is often determined by some constitutional disturbance, as, for instance, rickets, high arterial tension, an acute specific fever, pregnancy.

On examining the fundus, the choroidal coat seems the main tunic at fault; the change usually commences as an atrophy of the choroid which exposes the sclerotic to view, and so produces the appearance of a white or yellowish-white area extending to a variable extent from the optic disc. At first it is crescentic in shape, and confined to the outer border of the disc, as in fig. 1, opposite p. 203; the concavity of the crescent coincides with the edge of the disc, whilst its convexity projects towards the yellow spot; generally its border presents several patches of pigment. Later, this crescent becomes irregular in shape, increases in size, and so extends farther towards the yellow spot, and may surround the disc; very frequently associated with this condition are isolated patches of choroidal atrophy (see fig. 2). In its early stage it is called a *myopic crescent*, although a similar appearance is occasionally seen below the

disc in emmetropic or hypermetropic eyes—*congenital crescent* (see p. 229); later, it becomes conical in shape—*myopic conus*—the apex of the cone pointing towards the macula; in its more fully developed condition it always indicates that a considerable bulging (*posterior staphyloma*) has taken place at the posterior pole of the eye. We can often see the effects of successive outbreaks of the disease by the appearance of the staphyloma, which then presents several secondary crescentic edges, each being less white than the first.

The above view of the anatomy of the myopic crescent is not universally held. Some consider that, owing to the obliquity of the ocular end of the optic nerve and its canal in a highly myopic eye, the lamina cribrosa or the outer wall of the canal is visible through the transparent papilla in the form of a crescent.

The macular region shows very definite and characteristic changes. In an early progressive myopia there is usually a mottled appearance produced by very fine changes of a pigimentary and atrophic character, together with, frequently, several minute hæmorrhages. At the macula itself there is often seen a coal-black spot, which enlarges somewhat as the disease itself. Fine striations are sometimes present, probably fissures in the choroidal coat. Later, extensive choroidal atrophy with a large amount of proliferated pigment may involve the whole macular region. Many other pathological changes may be met with in myopic eyes, such as detachment of the retina, retinal hæmorrhages; general thinning of the choroid, which possesses little pigment, so that a reticulated vascular groundwork is seen beneath the retinal vessels, which are straighter than normal and increased over the temporal portion of the fundus; the lens may become dislocated, an attempt of Nature to rectify the optical defect of the eye, or show irregular opacities. The vitreous is usually more fluid than natural, often contains floating membranous opacities, and may be posteriorly detached.

A myopic eye presents other peculiarities besides those already mentioned; thus, the anterior chamber is frequently of great depth, the ciliary muscle is elongated, its transverse fibres being defective. The optic nerve enters the sclerotic obliquely, and the white matter

of Schwann often extends to the level of the retina over the whole disc, so that the physiological cupping is absent. The nerve-sheath, instead of ceasing at the point where the nerve enters the sclerotic, is prolonged a short distance into its substance, a condition which must still further weaken this part of the eye. The angle made by the visual line and the optic axis is smaller than in emmetropia; indeed, in some cases the visual line lies on the outer side of the optic axis, and the angle κ (see p. 465) is then said to be *negative*, producing an apparent convergent strabismus (see p. 559).

The subjects of these changes at the posterior pole of the eye are sometimes able to see very well when the proper correcting glass is used. When, however, there is a posterior staphyloma, and especially when this is progressive, the vision is almost invariably diminished to a great extent. When the yellow spot is actually involved, we of course find that all central vision is lost. The patient can then see only large objects, and to effect this he is obliged to rotate the head or the eyes to one side, so that rays from the object may fall on the peripheral parts of the retina.

Progressive myopia is pathological, and dependent on organic changes, whereas simple myopia of low degrees may be taken as a further development of the eyeball, fitting it for some higher and special purpose.

Though these two chief varieties have alone been described, many other intervening grades are met with, which cannot properly be classed under either heading.

When myopia has once become established, some of the conditions which combined to produce it are removed; thus the accommodation is used less, or not at all, and, since accommodation and convergence are associated acts, the myope finds it easier to give up convergence, and to use one eye only at a time for near vision. The elliptical shape assumed by the eyeball is less adapted for rotation within Tenon's capsule than the more spherical form of the emmetropic eye, and this also renders convergence difficult. In a short time the power of convergence becomes so impaired that it can be maintained, even by an effort, only for a few seconds, and before long may be altogether lost, and one eye remain in a state of divergence. (See Divergent Strabismus, p. 579.)

The action of the ciliary muscle and internal recti having been in this way annulled, the myopia may in favourable cases remain stationary; such eyes are, however, liable to suffer from an increase of their myopia if the general health is in any way impaired; and patients—mostly women—are not unfrequently met with who, having had a high degree of myopia since childhood, suffer after middle life without any obvious cause from its rapid increase, with atrophic changes in the choroid, the appearance of opacities in the vitreous, which is unduly fluid, and in the lens.

Apparent myopia.—A person may be emmetropic or hypermetropic, yet by subjective, and even objective, tests appear to be myopic. This is due to tonic spasm of the ciliary muscle, which does not relax in the dark room or under a convex lens. It is very apt to mislead a novice. The near vision is good, distant vision bad; the real nature of the condition is revealed only by thoroughly paralysing the accommodation by the use of atropine. It is seen more often in hypermetropes, occasionally in emmetropes, and rarely in myopes with error of low degree, giving it an appearance of being much greater than it really is.

Treatment of myopia.—The treatment of myopia may be best considered under the headings Prophylactic and General Treatment, Treatment by Glasses, and Operative Treatment.

Prophylactic and general treatment.—Since myopia is an acquired condition, its prophylactic treatment is of extreme importance, and its consideration is almost identical with the consideration of the proper hygienic surroundings in which the young should be brought up. School hygiene has received considerable attention of late years—buildings have been improved, furniture has become more rational, printing and paper are more suitable. It is very important that the illumination should be good; the children should on no account face a strong light, which for writing should come by preference from behind and to the left side. If artificial light is necessary, electric or incandescent gas light is the best, with a frosted globe; this should either be shaded or placed close to the ceiling of the room. The relative heights of the desks and seats should be so arranged that no stooping is necessary;

there should be a slight slope on the desks. For reading, the type should be clear, not too small, and the words and lines well spaced; the paper should not be glazed.

All cases of myopia, however slight, need careful treatment, since they are all liable to increase in degree, and may become progressive. Apart from the use of glasses (see below), strict attention to general health, hygienic surroundings, and regular exercise must be paid, and the amount of reading curtailed, until it has been ascertained that the myopia is not increasing. For this purpose the child should be examined again in from six to eight months' time.

If the myopia is of the active or progressive form, all work must be stopped. No reading or writing is to be permitted, all violent exercise must be prohibited, and any constipation must be met by gentle purgatives, since all straining is very harmful. At the same time the patient must have plenty of fresh air and good food, and may indulge in gentle exercise—which, indeed, is beneficial. Only a minimum amount of alcohol must be allowed. The eyes should be constantly under observation; and when the myopia has become stationary, change of air—a sea voyage if possible—should be prescribed.

Treatment by glasses.—This subject will be considered in a later section (see p. 546).

Operative treatment.—Removal of the crystalline lens for high myopia seems to have been performed before 1776, since it is mentioned in the writings of the Abbé Desmonceaux, published that year; it was long, however, before the treatment became at all general. The first actually recorded case was that published by Adolph Weber in 1858. Donders and von Graefe, however, condemned the treatment, which consequently languished until the time of Fukala, who performed his first operation in 1887 and published his first paper on the subject in 1890. The method he employed was that of repeated discissions, sometimes combined with an iridectomy. In his second paper, in 1894, he states that he has given up the iridectomy, and follows up his discission with an evacuation. The operation has been fully discussed by the Ophthalmological Society of the United Kingdom, and also by the French

Ophthalmological Society, to whose 'Transactions' the reader is referred.

Indications for the operation.—As a rule the operation should not be undertaken unless there are at least 14 dioptries of myopia if the patient is an adult, 10 dioptries if a child. Rapid increase of the myopia in a child may, however, necessitate the performance of the operation before this degree is reached. Inability on the part of the patient to do his or her ordinary occupation, other things being favourable, is a strong indication; especially is this the case with regard to domestic servants.

Contra-indications.—The operation should not be attempted if the vitreous is in a condition of synchysis; if the tension of the globe is markedly diminished; if the macular degenerative changes are extensive, although even in apparently hopeless cases improvement in central vision may take place; if there is any intra-ocular hæmorrhage; if extensive corneal nebulae are present; if the conjunctiva or lachrymal apparatus is unhealthy; or if the other eye has been lost, from whatever cause.

The operation.—In children and young adults the operation closely resembles that for lamellar cataract. The pupil of the eye having been dilated with atropine, the anterior capsule of the lens is freely divided with a cataract needle. In from three to seven days, or immediately any increased tension is detected (the eye in the interval being kept under the full influence of atropine), an evacuation of the opaque lens substance should be performed, either with a curette or by suction. A final discission of the lens capsule is in nearly all cases needed after from two to three months. After the age of thirty-five it is usually necessary to extract the clear lens, the method employed being exactly similar to that for cataract extraction.

Dangers, near and remote, following the operation.—The following complications and sequelæ may arise: glaucoma, irido-cyclitis, septic infection, prolapse of iris, anterior synechia, retinal and choroidal hæmorrhage, and retinal detachment. Nothing further need be said about these conditions, with the single exception of the last. Retinal detachment is

liable to occur in myopia itself, and it is extremely difficult to determine whether operative treatment increases or diminishes this liability. On the whole, evidence goes to show that a myopic eye after operation is not more prone to acquire retinal detachment than before operation. Some go so far as to say that the removal of the lens is prophylactic against detachment.

Optical result of the operation.—This varies with the degree of myopia present. Various formulæ have been devised, none of which are found practically to be accurate. Broadly, it may be stated that an eye with 22 dioptries of myopia, in an aphakic condition, becomes emmetropic; 18 dioptries of myopia are converted into 2 dioptries of hypermetropia; 15 dioptries into 3 dioptries of hypermetropia; and 10 dioptries into 5 dioptries of hypermetropia. But these numbers are by no means constant. The visual acuity is raised on account of the light which enters the eye being increased in intensity and diminished in the amount of its dispersion, and also because the retinal image is enlarged. It is impossible to state definitely whether the progress of the myopia is arrested by the operation; sufficient time has not yet elapsed to make any statistics on this point reliable.

(ii) **Hypermetropia** (H, fig. 122, and fig. 133). DEF.—*Hypermetropia is that condition in which parallel rays entering*

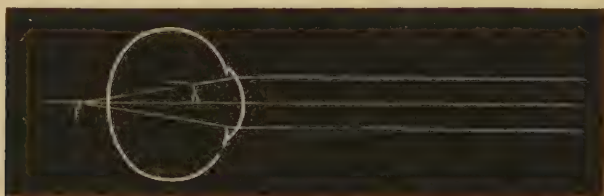


FIG. 133.—Parallel Rays entering a Hypermetropic Eye.

an eye at rest are so refracted that they do not come to a focus on the retina, but, if produced, behind it—i.e. the retina lies in front of the principal focus of the dioptric system of the eye. It may be due to the antero-posterior axis of the eye being too short—axial hypermetropia—and this is the common form; or to the refractive power of the eye being

diminished by flattening of the cornea, diminished refractive power in the lens, or absence of the lens—aphakia.

Since the retina lies in front of the principal focus, rays from any point on it will be divergent on leaving the eye (fig. 133),¹ and the conjugate focus of any such point will therefore be behind the eye, at the point where the diverging rays would meet if prolonged backwards. The *punctum remotum* (fig. 134) is therefore *negative* and *virtual*. As in myopia, an image of the fundus is formed at the far-point, but in this case the image is erect and virtual.

Since the rays from the retina are divergent on leaving the eye, it is evident that only convergent rays can be focussed on the retina; but in nature there are no such rays, hence a

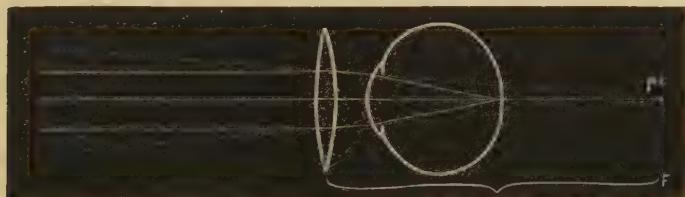


FIG. 134.—Parallel Rays entering a Hypermetropic Eye through a Convex Lens.

hypermetrope, with his accommodation relaxed, has no distinct vision of any objects, but by means of the accommodation the refractive power of the eye can be increased, and parallel and even divergent rays be brought to a focus on the retina if the ciliary muscle is sufficiently powerful. The muscular effort required will, however, be great in proportion to the nearness of the object, so that such eyes tire comparatively soon, if continuously fixed on near objects, while more distant ones may be viewed for a considerable time without fatigue; hence the popular term 'long-sightedness.' This condition must not be confounded with that of presbyopia, in which the refraction may be normal, but, the accommodation having become weakened from age, near vision is impaired.

We have seen that converging rays are the only ones which can be focussed by a hypermetropic eye with its accom-

¹ Compare also figs. 109 and 113.

modation at rest, and that there are in nature no such rays; parallel or divergent rays may, however, be rendered convergent by a convex lens. Thus if an eye were hypermetropic to such an extent that its far-point was 20 cm. behind the cornea, a lens of such a strength, and so placed, that its principal focus coincided with this point, would give to parallel rays the required amount of convergence, and cause them to come to a focus on the retina without any accommodation being used, so that such an eye, with its hypermetropia corrected in this way, would see distant objects under the same conditions as the emmetropic eye (fig. 134).

Since the defective vision due to hypermetropia can be obviated by the use of the accommodation, a small amount may exist without causing any symptoms; if, however, a hypermetrope is called upon to use the eyes much for near objects, trouble, varying in degree and kind in different individuals, is experienced. In slight cases the eyes become tired and bloodshot after being used for some hours. In others the work or book has to be laid aside after a few minutes, owing to the sight becoming dim, or the eyes filling with tears—a group of symptoms often classed under the name of *accommodative asthenopia*. In others, again, reading is always followed by headache, which occasionally is so severe that it is attributed to cerebral causes, and the subject of it condemned to spend his or her time in idleness, when the whole trouble might be removed by correcting the hypermetropia with suitable glasses.

Hypermetropia is a congenital defect due to an imperfect development of the eyeball during the first few years of life, but it is seldom discovered until the child begins to learn to read. At birth all eyes are hypermetropic, and, as a rule, the emmetropic condition is not reached until the age of five or six. Any lack of development during this period produces permanent hypermetropia. The symptoms in children differ somewhat from those met with in the adult. Often one of the first indications of there being anything wrong is that the child holds the book very close to the face, and is therefore supposed to be short-sighted. Myopia, however, in young children is rare, and the presence of hypermetropia under

these circumstances should always be suspected. The myope obtains clear images of the minutest objects if held within his range of vision, and therefore reads the smallest type with ease; the hypermetrope, on the other hand, can obtain clear retinal images only by using his accommodation, and the nearer the object, the greater is the effort required; but the size of the retinal image increases in proportion as the distance is decreased, and increases at a greater ratio than the circles of diffusion caused by imperfect focussing; hence the child will sometimes prefer to hold the book so near that the ciliary muscle is unequal to the exertion necessary to focus the rays on the retina, because by that means he obtains a large image with less muscular effort than if he held it at a distance for which his accommodation was sufficient.

In other cases the efforts made by the ciliary muscle to respond to the call made upon it result in the production of a tonic contraction, or *cramp*, by which the eye is maintained in a condition of accommodation for a near point—*apparent myopia* (see p. 495).

In many instances of hypermetropia, generally of moderate degree, the accommodation is equal to the necessary effort only when it has *convergence* associated with it; hence the child (these cases mostly occur in children) suffers from no defect of vision, but develops a *convergent strabismus*. This subject will be considered more fully in a subsequent chapter; it is sufficient here to note the fact that a greater amount of accommodation can be used if convergence is associated with it than if used alone; and that convergent strabismus in a child is an almost certain sign of hypermetropia.

(iii) **A. Regular Astigmatism** (fig. 135). DEF.—*Astigmatism is that condition in which rays entering the eye are refracted unequally in different meridians.* In considering optical principles and the laws of refraction, we saw that rays from any point, being refracted at a spherical surface, again came to a focus, and formed an image of the point. If, however, one meridian of the refracting surface had a different curvature from the others, it is evident that their focal distances would also be different, and that the rays would therefore no longer all be focussed at one point. Such a surface is there-

fore said to be astigmatic. A familiar example of an astigmatic surface is the bowl of a spoon.

In surfaces which are regularly astigmatic—which the bowl of a spoon is not—the various meridians have the same curve throughout. Those having the longest and shortest radius of curvature are called the principal meridians, and are always at right angles to each other.

It will be necessary to consider in detail the action of an astigmatic surface. In fig. 135 let rays from a point f fall on an astigmatic surface $acbd$, and let the conjugate focus of f be at f'_2 for rays which pass through the vertical meridian ab , and at f'_1 for those which pass through the horizontal meridian cd ; it is evident that the section of the cone of rays after

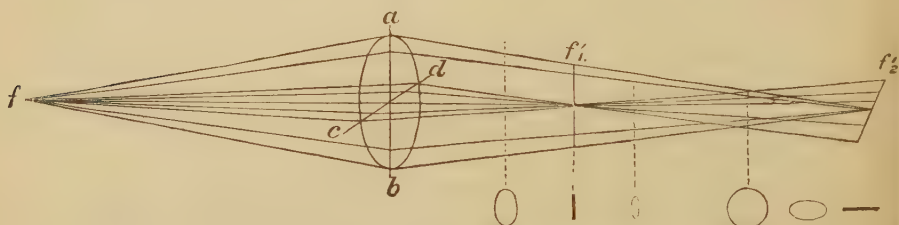


FIG. 135.—The Course of Rays in Astigmatism.

refraction will vary in shape according to the position at which it is made; thus, between the refracting surface and f'_1 it will be an oval diminishing in size towards f'_1 , the horizontal meridian shortening more rapidly than the vertical; so that as we approach f'_1 we get an oval gradually becoming narrower, until at f'_1 the section is indistinguishable from a vertical line; between f'_1 and f'_2 the vertical diameter will continue to diminish, while the transverse will increase, so that we obtain successively an oval with a long vertical diameter, a circle, an oval with long transverse diameter, and at f'_2 a transverse line. Hence we get this rule:

If rays from a point are refracted by an astigmatic surface, a linear image of the point is formed at the focus of each principal meridian; and the direction of the line is at right angles to that of the meridian at the focus of which it is formed.

If the above rule be kept in mind, all the phenomena of refraction which occur in an astigmatic eye will be readily understood.

Although we have spoken of the cornea as a spherical surface, it is rarely strictly so ; usually its vertical meridian has a somewhat shorter radius of curvature, and therefore a greater power of refraction than the horizontal, so that most eyes are astigmatic in a very slight degree ; it is only, however, when the difference between the principal meridians is sufficient to interfere with vision that the defect comes under the notice of the surgeon. Astigmatism of the cornea may be increased, diminished, or neutralised by a similar condition in the crystalline lens.

Astigmatism is, then, the condition in which the eye refracts differently in its different meridians. It is usual to classify regular astigmatism into five varieties, which are enumerated in the accompanying table, and this arrangement is a convenient one. It should be borne in mind, however, that the difference between these does not really consist in a difference in the nature of the astigmatism, but in the difference in the refraction of the eye when the astigmatism has been corrected by rendering the principal meridians equal by an alteration in the refraction of one of them.

Variety of regular astigmatism	Refraction of the principal meridians	Condition to which the eye may be brought by correcting the astigmatism
1. Simple myopic . . .	{ Emmetropic Myopic } MAs	} Emmetropia
2. Simple hypermetropic	{ Emmetropic Hypermetropic } HAs	
3. Compound myopic .	Both myopic MMAs	Myopia
4. Compound hypermetropic	Both hypermetropic } HHAs	Hypermetropia
5. Mixed	{ Myopic Hypermetropic } MHAs	{ Myopia or hypermetropia according to which meridian is corrected

In simple astigmatism the retina lies at the focus of one of the principal meridians, and the retinal image of a point will therefore be a line at right angles to that meridian. This can

be impressed on the memory by a simple experiment. Let the reader render his own eye astigmatic by placing a cylindrical lens in front of it; the axis of the cylinder, shown by marks on the glass, will then be the direction of the unaltered, or emmetropic, meridian; if, now, a point of light be looked at, obtained by looking at a pinhole aperture in a card held close to a flame, the point will be seen as a line of light; and in whatever position the lens is held, the direction of the line will always be at right angles to the axis of the cylinder—*i.e.* to the emmetropic meridian.

If now, under the same conditions, a straight line be looked at, it will be found that it is seen clearly only when its direction is at right angles to the emmetropic meridian; this is because every point of the line is seen as a minute line, and may be explained as follows: If, instead of a luminous point

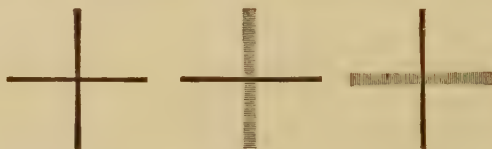


FIG. 136.—Cross-lines as seen by an Emmetropic Eye, and by two Astigmatic Eyes.

as is seen by looking at a pinhole aperture in front of a light in the manner just described, a black point or dot be seen, it will, to an astigmatic person, appear to be elongated into a linear image parallel to the ametropic meridian. A line is merely a succession of points or dots, so close together as to be fused into a linear object; so, if astigmatism exists in the horizontal meridian, each of these dots, the elements of a line, appears linear horizontally, and a horizontal line would appear black and clearly defined, owing to the overlapping of the linear diffusions of its component elements; a vertical line, however, will show these linear diffusions, and will consequently appear blurred and ill-defined (see fig. 136).

Hence the following rule:

An eye with simple astigmatism (one of the principal meridians emmetropic) can see clearly only lines whose direction is at right angles to its emmetropic meridian.

B. Irregular astigmatism exists in a very slight degree under normal circumstances in most, if not in all, eyes, from lenticular defect. The lens-fibres are the cause of this slight aberration, and give rise to the apparent existence of radii to stars. If a star be looked at through a small aperture, not more than half a millimetre in diameter, no radii will be visible. This defect does not cause any inconvenience in ordinary vision. It is, however, exaggerated in incipient cataract, causing visual disturbance and much mental perturbation. Irregular astigmatism, dependent upon conical cornea or previous corneal ulceration, is a source of great annoyance and visual defect, and cannot be remedied by the use of glasses. External objects form distorted images on the fundus, and are perceived as such. Looking through a badly made pane of glass, distortion of trees and other objects is noticed; this is the condition perceived by an eye with irregular astigmatism, only to a greater degree.

Asthenopia, or subjective signs attending ametropia, &c.—It is advisable to make a few remarks about the symptoms of refractive errors and defective accommodation. The patient nearly always complains of *frontal headache* or pain at the back of the eyes; *inability to read or to do near work* for any length of time without the words running into one another, or the objects becoming blurred; and the eyes are said to water, smart, and become red—*conjunctival congestion*. These three constitute the most marked symptoms, and suggest a prompt attention to the refractive condition of the eye. The cause of asthenopia is due to the associated acts, convergence and accommodation, being in some way interfered with. In myopia, convergence seems the function at fault; in hypermetropia and presbyopia, accommodation appears to produce these symptoms; therefore, the former has been called *muscular asthenopia*, and the latter *accommodative asthenopia*. But the symptoms are not produced in either case by a fatigue of one function, but by *the difficulty of working the two together*, owing to the disturbance wrought by the ametropia upon their association (see p. 483). It is a well-known fact that these symptoms disappear when the error is corrected or when a concomitant squint develops. In the

former case, the proper association of these functions is restored ; in the latter, it is broken, *i.e.* there is a complete functional disassociation.

If there are different refractions in the two eyes—*anisometropia*—for example, myopia in one, and hypermetropia in the other, or different forms of astigmatism—the ametropic symptoms are usually pronounced.

Section V.—LENSES USED IN TESTING REFRACTION.

THE OPHTHALMOSCOPE.

(i) **Trial Lenses.**—Before describing the various methods of ascertaining the refraction of an eye, it will be necessary to make a digression in order to explain the principles on which the lenses used for this purpose are numbered, and to explain the nature and use of the ophthalmoscope, which is also used for the same purpose.

In this country there are two modes in use of numbering lenses.

The one is to give to each lens a number expressing its focal length in inches ; thus, we speak of a 3-in. or 6-in. lens. There are several objections to this method : in the first place, since the strength of the lens is in inverse proportion to its focal length, it is necessary to invert the numbers in order to make them represent the relative power of the lenses ; so that in calculating the power of a lens we should speak of it as a $\frac{1}{3}$ rd or $\frac{1}{6}$ th ; this becomes inconvenient when several have to be added or subtracted. Then, in the higher powers the intervals between the lenses are necessarily unequal. Another great drawback to the system lies in the fact that the inch has a different value in different countries ; and as many opticians use foreign glasses, it is not always clear what is meant by a particular number.¹

The other system of numbering lenses is based on the metrical system of measurement, and is now in very general use. The unit is a glass of a metre focal length, and this is termed a dioptré (1 D) ; all other lenses are enumerated as fractions or multiples of this ; thus, a lens having a focus of

¹ 1 English inch = 25·3 mm. 1 Paris inch = 27·07 mm.

two metres would be half this strength, and would therefore be 0.5 D, while one having a focus of half a metre would be 2.0 D, a third of a metre 3.0 D, and so on. The focal length of any lens numbered on this system is found by dividing a metre by the number of the lens; thus, 4.0 D would have a focal length of 25 cm., or 10 English inches.¹

A case of trial lenses should contain pairs of convex and concave spherical lenses from 0.25 D to 20.0 D, and cylindrical lenses convex and concave from 0.25 D to 6.0 D.

Spherical lenses have been already sufficiently described, but a few words of explanation are necessary as to the nature of the cylindrical. One surface of such a lens is, as the name implies, a portion of a cylindrical surface; the other is usually plane. If from a portion *cdef* (fig. 137) of a glass cylinder *AB*, of which *ab* is the axis, a circular piece be cut out, its vertical diameter *hi*, parallel to the axis *ab* of the cylinder, will be equal in thickness throughout its whole length, and cannot, therefore, alter the relationship of parallel rays; it is called



FIG. 137.—A Glass Cylinder.

the axis of the lens. The horizontal meridian *kl* will be thicker at its centre than at its extremities, being in section plano-convex, and will have the power of bringing parallel rays to a focus. A circle one inch and a half in diameter cut out of such a portion of a glass cylinder would constitute a convex cylindrical lens (+ cyl.). Assuming the posterior half of the glass cylinder to be hollow as at *H*, it is easy to conceive that a plano-concave cylindrical lens (− cyl.) could be obtained in a similar manner;

¹ The relation of centimetres to inches is approximately as 5 to 2; one centimetre being equal to 0.3937 inch.

the plane meridian would still coincide with the axis of the cylinder, while the most concave would be at right angles to it. The direction of the axis of the cylinder is marked on the glass either by two lines, one at each side, or by a portion of the lens on each side being ground with the edges of the ground portions parallel to the axis; this meridian, being plane, has no refractive power. The lens is numbered in accordance with the refraction of the meridian of curvature—*i.e.* the one which is at right angles to the axis; thus, a cylindrical lens of

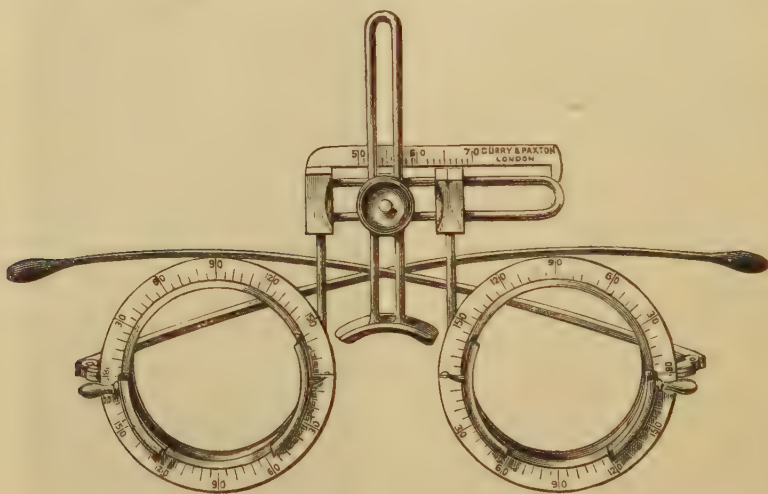


FIG. 138.—Tosswill's Adjustable Trial Frame.

6 dioptries (6 D cyl.) means that the refracting power of the meridian of curvature is equal to that of a lens of 6 dioptries.

From the nature of a cylindrical lens, it follows that the addition of one to an eye which is not astigmatic would render it so, and that by one of suitable strength the difference between the principal meridians of an astigmatic eye could be neutralised and the astigmatism corrected.

Besides lenses, a trial case should contain a set of prisms, numbered according to their angles from 1° to 12° , an adjustable trial frame (fig. 138), a block of the same size as the lenses to occlude one eye, a slit 1 mm. wide, also mounted like a lens, and a few diaphragms and coloured glasses.

(ii) **The Ophthalmoscope.**—As the ophthalmoscope affords one of the most useful means which we possess of testing refraction, it is necessary to say a few words here concerning its construction and use. The rays which come from any point on the retina of an emmetropic eye leave the eye in a state of parallelism (fig. 119), and could therefore be focussed on the retina of another emmetropic eye. But the only light which comes from an eye is the reflected portion of that which has entered it through the pupil; and, since the emerging pencil follows the same course as that which entered the eye, it follows that the observer's head cannot be placed in a position to receive the former without at the same time intercepting the latter. The ophthalmoscope is a contrivance for throwing light into the eye, and allowing some of the returning rays to enter the observer's eye. It consists essentially of a mirror, which, while reflecting some rays, transmits others.

The original ophthalmoscope, invented by Helmholtz in 1851, consisted of three parallel plates of glass, separated from each other by small intervals; by means of this, held at a suitable angle, light from a lamp was reflected into the eye, and of the light which returned from the fundus some was reflected from the glass and lost, but a portion was transmitted through it to the observer's eye; and, being focussed on its retina, produced an image of the fundus of the eye under examination. Helmholtz's ophthalmoscope can be used with less discomfort to the patient than perhaps any other form; but the illumination of the fundus obtained by it is much less than with the more modern instruments, and it requires considerable practice to use it with ease. It was soon found that the fears originally entertained of damage being done to the eye by exposure to light were not well founded, and instruments were accordingly constructed in which the mirror was made of polished metal or silvered glass, a central perforation allowing the passage of some of the returning rays.

The modern ophthalmoscope consists essentially of a mirror, which may be plane or concave, having a small central aperture; and this is all that is necessary for the purpose for which the instrument was originally constructed—namely, that of seeing the fundus of the emmetropic eye. But for estimating

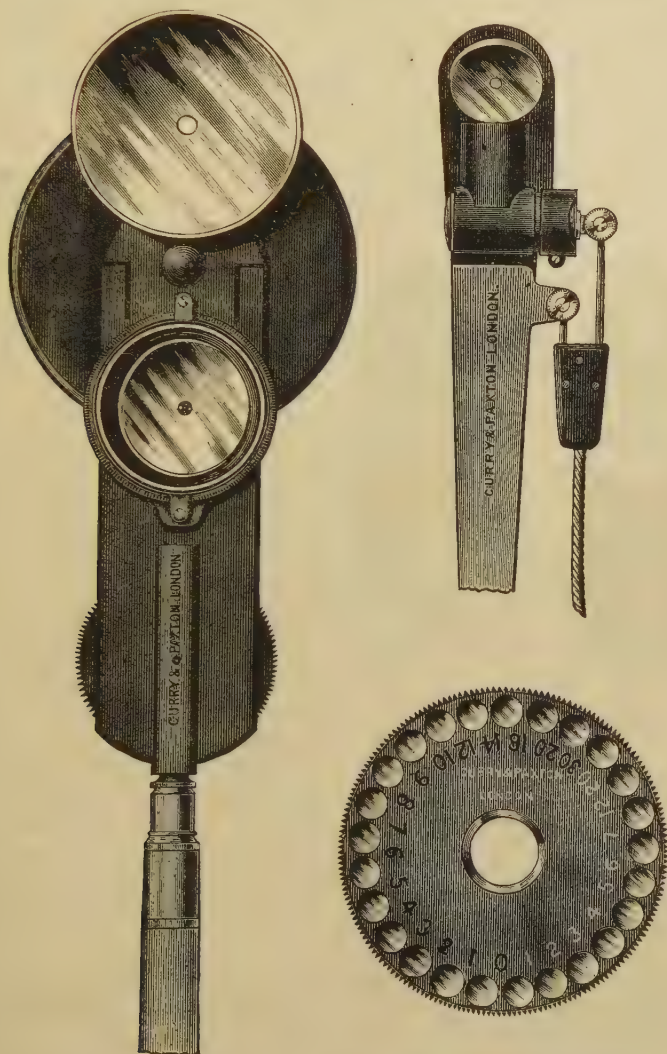
refraction, it is necessary to have an arrangement by which different lenses can be placed behind the sight-hole ; and it is chiefly in the mode in which this latter requirement has been fulfilled that the various instruments differ from each other. The variety of ophthalmoscopes is so great that a mere enumeration of them would occupy a considerable space, and serve no useful purpose ; it will suffice to indicate the conditions which are essential to a good instrument.

The mirror, if there is only one, should be concave, have a focal length of not less than 22 cm. (9 inches), and a diameter of not less than 1 inch. A second smaller mirror set obliquely with a focal length of about 2 inches is an advantage, but not essential, and a plane mirror is often useful. There must be a series of convex and concave lenses, and an arrangement by which these can be successively placed behind the sight-hole without removing the instrument from the eye. Much difference of opinion exists as to the number of lenses necessary. Couper, who was one of the earliest to use the ophthalmoscope systematically for the estimation of refraction, considers that every power should be obtained by a single lens, and that combinations are inadmissible ; as he also considers that every ophthalmoscope should possess a lens sufficiently powerful to correct the highest degree of myopia which is likely to be met with, his ophthalmoscope necessarily contains a very large number of lenses.

A suitable instrument for refraction and ophthalmoscopic examination is represented in fig. 139. It consists of a disc containing two series of spherical lenses : one convex, 1, 2, 3, 4, 5, 6, 7, 12, and 20 dioptries ; the other concave, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 12, 14, 16, 20, and 30 dioptries. The disc, shown separately in the figure, is milled at the edge, and is made to revolve to the right or left by means of two other milled discs, one of which is worked by the index finger of the hand holding the instrument ; thus, any lens required may be placed in the sight-hole of the instrument. A sector may be connected to the back to carry any other lenses required. The strength of the lens is shown in a small circular aperture at the back : white figures indicate concave lenses ; red figures convex lenses.

Three different mirrors may be used : a small one of 8 cm. focal length, set at an angle of about 30° , which can be revolved upon its

central axis by means of a milled edge ; and two large mirrors, plane and concave, which are reversible upon a horizontal axis by means



FIGS. 139 and 140.—Juler's Ophthalmoscope and Electric Ophthalmoscope.

of a spring clip. The large concave mirror has a focal length of 30 cm. The mirrors are arranged on a metal plate fixed to the back-

piece by means of a screw situated equidistant from the central apertures of the small and large mirrors, so that by rotation the aperture of either mirror may be brought into position in front of the sight-hole of the ophthalmoscope. The central aperture in the small mirror is 2 mm. in diameter; in the large, 4 mm.

The large mirrors are used for indirect ophthalmoscopy and retinoscopy, the small mirror for direct ophthalmoscopy.

To the right (fig. 140) is a drawing of an electric ophthalmoscope, which is suitable for the direct method only, and is so arranged that by the use of an electric light the fundus of any person

may be readily examined, even in broad daylight. The mirror is concave with a short focal length (5 cm.) ; it is set obliquely, its axis being directed downwards. Immediately below the mirror is fixed a small electric lamp, the rays of which are, by the reflection of the mirror, projected into the eye under examination. It can be adapted to any ordinary ophthalmoscope. It is very convenient when a suitable lamp or dark room is unobtainable. Its principal value, however, is made manifest in the examination of fundi in which the media are cloudy, whether it

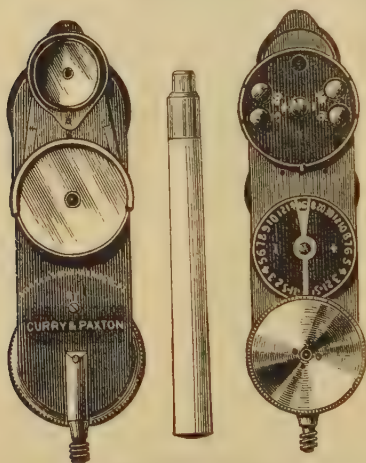


FIG. 141. Morton's Ophthalmoscope.

be lenticular opacity or a vitreous haze. In the latter the real cause may be made out when an ordinary ophthalmoscope cannot furnish any further information beyond the existence of the opacities. The light of this ophthalmoscope causes no greater annoyance to the person under examination than the ordinary instrument, since, by means of a resistance coil, the light can be graduated to any intensity.

Fig. 141 represents the excellent and largely used ophthalmoscope devised by Morton; it has considerably more lenses than the previous instrument.

There are two methods of using the ophthalmoscope—the *direct* and the *indirect*. Each of these has advantages of its own, and neither of them should be practised to the exclusion of the other. Before passing to a detailed description of these,

there are certain practical points common to both of them which must claim our attention.

In the first place, an artificial source of light is necessary ; sunlight, it is true, can be employed, but there are obvious inconveniences which practically preclude its use. Any steady broad flame will answer the purpose, a circular gas-burner, such as an Argand, or an electric lamp with a half frosted globe, being the best. It is convenient to have it affixed to a bracket, which allows of free movement both in a vertical and in a horizontal plane ; and a shade by which lateral rays can be arrested is sometimes useful. The examination should be conducted in a dark room. The lamp must be placed on the same horizontal plane as the eye, sufficiently far back to prevent any direct rays falling on the cornea, and only removed laterally a sufficient distance to avoid discomfort from its heat.



FIG. 142.—Direct Ophthalmoscopic Examination.

Before commencing to examine a patient, the knack should have been acquired of so manipulating the mirror as to throw the light on any required spot, and to keep it there during any movements of the head ; this is easily learnt with a little practice, but the want of that practice causes much disappointment at the first trial with the ophthalmoscope, and a considerable amount of discomfort to the patient.

In the *direct method* the surgeon sits facing the patient on the same side as the eye which is to be examined, in such a position that, when his face is brought close to the patient's, his own eye is opposite the same eye of the patient. The mirror, with the observer's eye close behind the sight-hole, is held close to the patient's eye (see fig. 142). If the relative positions of surgeon and patient are such as have been described,

the former will naturally use the left eye for the patient's left, and *vice versa*. Holding the mirror as close as he can without losing the illumination, the surgeon looks through the sight-hole into the patient's eye; if this is emmetropic, the parallel rays from it enter his own eye and are focussed on his retina; he accordingly sees all the details of the patient's fundus. It is essential that neither patient nor surgeon should use any accommodation, for in the one case the rays would leave the patient's eye convergent instead of parallel, and in the other, although parallel, they would not be focussed on the retina of the surgeon. By this method the details of the fundus are

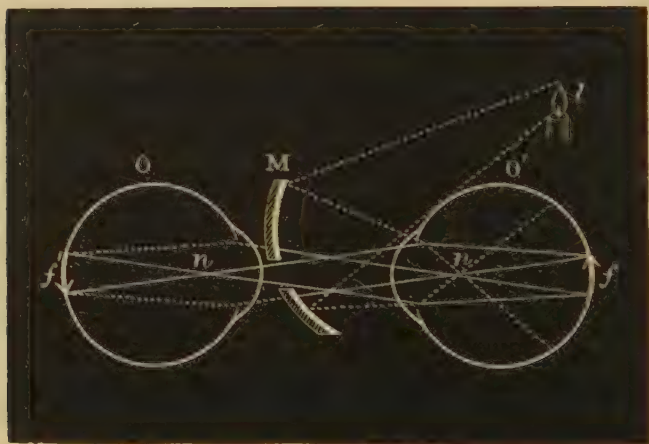


FIG. 143.—The Course of Rays in Direct Ophthalmoscopy.

seen in their true position, but magnified about fourteen times by the dioptric system of the eye. The image is therefore erect.

In fig. 143, which illustrates the course of the rays in direct ophthalmoscopy, part of the illuminated area of the fundus f of the patient o' is seen by the observer o . The details of the fundus of o' are inverted on the fundus of o , and so are perceived erect.

In the *indirect method*, the ophthalmoscope or mirror is held at a distance of little over half a metre from the patient; at the same time a convex lens is held close in front of the eye

to convert the emerging rays from the fundus into an inverted aerial image (see fig. 144). If the eye is emmetropic, the rays, being parallel, will come to a focus at the principal focus of the lens. In ametropia, the image will be formed sooner or later according to the nature of the error.

The nearer this inverted aerial image is to the eye of the observer, the larger it appears, because it subtends a greater visual angle; so, by increasing the distance between the lens and the patient's eye, a slight increase in the size of the image is perceived; at the same time, a smaller area of the fundus is visible, owing to the di-

verging pencil of rays escaping beyond the periphery of the lens. Hence, the weaker the lens used, the larger the image will appear without loss of the surrounding details. This gradual enlargement of the image by the movement of the lens is so slight in emmetropia, that, for all practical purposes, it



FIG. 144.—Indirect Ophthalmoscopic Examination.

may be considered *nil*. In myopia, the image appears to enlarge rapidly; in hypermetropia it diminishes in size. A convenient lens for indirect ophthalmoscopy is one of 13 D (3 in.); those sold with the ophthalmoscope have sometimes a longer focus than this, and do not magnify sufficiently. The larger its diameter, within convenient limits, the better; a good size for the pocket is 2 in. diameter. The image obtained by this method is *inverted* and *real*, while the direct gives an *erect* and *virtual* image. By the indirect method a large portion of the fundus, magnified about four times, can be seen at one time, and it is therefore the better one for obtaining a general view of the fundus, and should be used first in every case. The direct gives more detailed information concerning a smaller area at one time.

In indirect ophthalmoscopy there is no real necessity to use

any lens in the ophthalmoscope, though many surgeons use + 2 D, + 3 D, or + 4 D for the following reasons: accommodation is necessary if no lens is used, and so a weak convex lens will relieve the accommodation. Those who are presbyopic, and those who are accustomed to relax their accommodation upon *direct* ophthalmoscopic examination, will unconsciously do so upon using the *indirect* method, and so will not see a clear image without a weak convex lens in the ophthalmoscope. Secondly, the aerial image of the fundus will be magnified by the addition of a weak convex lens.

In the direct method lenses must be used to neutralise any ametropia existing, either in the examiner or in the patient,

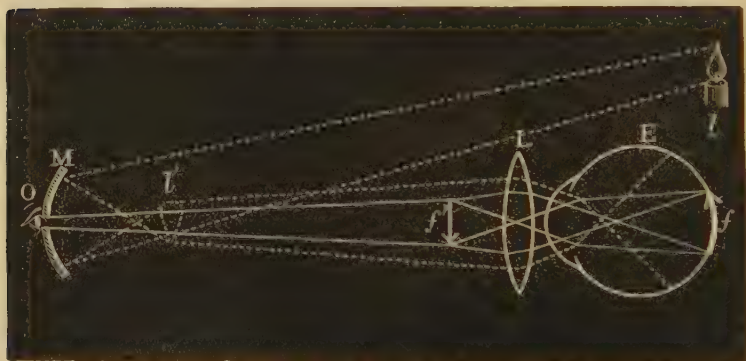


FIG. 145.—The Course of Rays in Indirect Ophthalmoscopy.

before a clear view of the fundus can be obtained with both eyes at rest.

The course the rays take in indirect examination of the fundus is seen in fig. 145.¹ The dotted lines illustrate the way in which the fundus is illuminated. The uninterrupted lines show the course of the rays from the details on the fundus of the person under examination to the eye of the observer: *l* is the luminous source, *m* the concave mirror, and *l'* the inverted aerial image of the flame; *E* is the eye under examination, *f* is a small portion of the illuminated area

¹ The inverted aerial image *f'* of the fundus should be several times the size of the object *f*, but for obvious reasons this is not shown in the figure, which is essentially diagrammatic.

of the fundus, which is converted by the lens L into an inverted aerial image f' , which is perceived by the observer, o .

There are several difficulties to be overcome in using the direct method: the first is that of getting sufficiently close to the eye without losing the illumination; this is, however, easily accomplished if the manipulation of the mirror has been previously learnt. If the patient's eye be directed straight forward, a difficulty sometimes arises from the reflection of the mirror being seen in the cornea; this is avoided if the eye be directed a little to the nasal side, and this position has the additional advantage that the posterior pole of the eye is rotated outwards, so that the optic disc, which lies slightly to the inner side, comes into view. We have seen that accommodation on the part of either surgeon or patient prevents a clear view being obtained of an emmetropic fundus; on the part of the patient this can generally be obviated by taking care that the other eye has no light falling on it, and that it is directed to a distant object. The surgeon's own accommodation is a more difficult matter to control. Some idea of the difficulty, and of the kind of effort required to overcome it, can be obtained by attempting to read through a convex lens a page of print placed at its focal distance; at first this will be found to be difficult, but the knack can be acquired with a little practice. When using the ophthalmoscope, it is well to try to imagine that one is looking at a distant object, and this is facilitated by the other eye being kept open. The small size of the pupil will not often prove an obstacle to the examination of the optic disc if the above precautions be adopted, but it frequently prevents a view being obtained of other parts of the fundus. The pupil contracts less if a plane mirror be used, and still less with an ophthalmoscope on the principle of Helmholtz's. The use of a mydriatic is, however, often necessary or advisable, for without one it is impossible to examine the extreme periphery of the fundus, and very difficult to obtain a good view of the macular region. The drugs that have been used for this purpose are sulphate of atropine, hydrobromate of homatropine, and a combination of the latter with hydrochlorate of cocaine. Of these the last is the best to use, since its action on accommodation (see p. 522) is less marked than the other

two. Cocaine alone in some cases produces partial dilatation of the pupil with little or no effect on accommodation, but its action is extremely uncertain. Lately, euphthalmine has been fairly extensively used as a mydriatic. Its usual action appears to be dilatation of the pupil in from thirty to sixty minutes, with only a slight and transitory effect on the ciliary muscle. Occasionally, however, its cycloplegic action is more marked than its mydriatic effect.

The small size of the pupil is a more serious obstacle to the indirect method, owing to the greater concentration of the light; and here the use of a mydriatic is more frequently necessary. The chief difficulty in this method lies in manipulating the lens and mirror at the same time. The best plan is to throw the light on the eye first with the mirror alone, then to interpose the lens, holding it at a little less than its own focal distance from the patient's eye; the head must then be moved backwards and forwards, care being taken not to lose the red reflex, until the details of the fundus are clearly seen. If any trouble arises from an image of the flame or mirror being seen reflected in the lens, a very slight rotation of it on its vertical axis throws the image out of the way.

In order to see any peripheral part of the fundus with the direct method, the patient must be told to look in the corresponding direction—*e.g.* upwards for the upper part of the fundus, downwards for the lower. With the indirect method, it should be borne in mind that the image moves in the same direction as the lens, and in the reverse direction to the surgeon's head; by a combined movement, therefore, in opposite directions, of lens and mirror, the part of the fundus which is visible may be changed at will.

Section VI.—METHODS OF ESTIMATING REFRACTION.

We are now in a position to consider the various modes of estimating the exact refraction of the eye. These are very numerous, but they mostly come under one of two heads: either they are *subjective* in character—that is, they depend on the visual sensations of the patient—or they are *objective*, and depend on what the surgeon himself observes. The

subjective methods for the most part, though not entirely, are founded on changes made in the patient's vision by glasses. Such a method has the obvious disadvantage that the results depend on the statements of the patient, who may be stupid or ignorant. On the other hand, with an intelligent subject it is often the quickest; and as the object of the examination is usually to ascertain what is the most suitable glass, its results are more appreciated by the patient. Some methods combine both the subjective and objective principles, and few surgeons care to rely upon either exclusively in a difficult case.

i. **Testing by trial lenses.**—At the outset the reader is again reminded that a perfectly emmetropic eye has clear retinal images of distant objects *without the use of any accommodation*, and a glass does not correct an ametropic eye (*i.e.* neutralise its ametropia) unless it places it in this condition.

If the reader has followed what has already been said concerning myopia and hypermetropia, he will often be able to form a correct opinion in a given case, from the patient's description of the symptoms, as to which of these errors is the more likely to be present; it will be better, however, for the present to disregard the symptoms altogether, and to suppose the diagnosis to be made entirely by means of the test-glasses.

The patient should be placed at a distance of 6 m. (20 ft.) from Snellen's test-types, and it should be ascertained what is the smallest line which can be read by the eye under examination—it is, of course, essential that the other eye should be covered—and the result should be noted in the manner described on p. 473. It is a good plan now to test the near vision with the reading-types, not because it is always, or even generally, essential for ascertaining the refraction, but because it may be required for this or for other purposes, and if not done at this stage is apt to be forgotten. In noting the near vision, the smallest type should be found which the patient can read, choosing his own distance, and then the farthest and the nearest point at which he can see it.

Example.— $R^1 V = \frac{6}{9}$ and 0.5 Sn.¹ 20–50 cm.²

¹ The letters R and L are used throughout to indicate the right and left eye respectively. Sn. indicates Snellen's reading-types, and J. those of Jaeger.

² It is more consistent with the plan on which these tests are arranged

If distant vision is found to be normal, it does not follow that the eye is emmetropic, unless it can be proved that no accommodation was used; *myopia, however, is excluded.*

The distant vision having been noted, a weak convex lens (+0.50 D) is placed before the eye; the subsequent steps of the test will depend on the effect which this has on vision; these will therefore be considered under two separate headings.

A. Vision is not rendered worse by a weak convex lens.

B. Vision is rendered worse by it.

A. If distant vision is not impaired by a convex lens, hypermetropia is present; for the effect of the lens is to render the parallel rays convergent, and only a hypermetropic eye can focus converging rays (vide p. 499). The strength of the lens should now be gradually increased until the strongest is found which the patient can bear without vision being made any worse; an amount of hypermetropia corresponding at the least to this must be present. The error thus discovered is called the manifest hypermetropia (M.H.).

Example.—Supposing that vision of $\frac{6}{9}$ is changed to $\frac{6}{6}$ by the addition of +1.5 D, and that a stronger glass impairs vision, the result is written thus:

$$L V = \frac{6}{9} \bar{c} + 1.50 D = \frac{6}{6},$$

therefore 1.50 = the manifest hypermetropia. In the same way, if vision remained the same with the addition of a convex glass, the glass would be the measure of the manifest hypermetropia, and the result might be written thus:

$$L V = \frac{6}{6} + 2 D = \text{M.H.}$$

We saw, however, on p. 500 that hypermetropia may be concealed by the action of the accommodation; and having found the *manifest* hypermetropia by the above method, we have no guarantee that a further amount does not still remain

that the metrical notation should be maintained throughout, but the result may be expressed in inches if preferred.

concealed by the accommodation. As a matter of fact, in young subjects this is usually the case, for, having constantly to accommodate in order to see, the act is performed instinctively as soon as an effort is made to look attentively at an object; and although, by a very gradual transition from the weaker to the stronger glasses, the accommodation can be coaxed to relax to a certain extent, some frequently remains in use concealing an unknown quantity of hypermetropia, which is therefore said to be *latent*. It is of course possible that the whole of the hypermetropia may be latent, so that the fact that a weak convex lens renders vision worse does not necessarily exclude the existence of hypermetropia. But it is rare, except in children, for all the hypermetropia to be latent; in patients over thirty, on the other hand, it is unusual for any to be latent. If the patient's 'near-point' is farther away than it should be at his age (see table, p. 478), hypermetropia may be suspected to exist, although none may be manifest.

Not only may the action of the ciliary muscle entirely conceal the existence of hypermetropia, but it sometimes passes into a condition of tonic contraction in excess of that required for distant vision, so that the eye is maintained in a condition of accommodation for a near point. As this spasmodic contraction cannot be voluntarily relaxed, the eye appears to be short-sighted. This *spasm of the accommodation* undergoes a partial, and sometimes a complete, relaxation in the dark, so that by examination with the ophthalmoscope in the dark room the apparent myopia (see p. 495) may be proved to be fictitious, or the existence of hypermetropia be diagnosed.

In order to ascertain with certainty the amount of latent hypermetropia it is necessary to paralyse the accommodation. There are several agents, called cycloplegics, by which this can be temporarily accomplished. The commonest of these is *sulphate of atropine*. A solution in water of the strength of 0.5 per cent. (2 gr. to the oz.) is the best for the purpose, and it should be dropped into the eye, if complete paralysis of the accommodation is required, three times a day for about three days. In young children, owing to the greater strength of the accommodation in them, it is often necessary to use it for a week or more. The final application should always be made

about an hour before the examination of the eye. In addition to the paralysis of the accommodation the pupil is widely dilated, and the effect does not fully pass off for a week, ten days, or even longer, after the last application.

Owing to the serious inconvenience that a patient suffers from the slow recovery of the function of accommodation after atropine, other agents have been employed as cycloplegics whose action is less lasting. The sulphates of daturin and duboisin act efficiently, but, although the effect lasts only about half as long as that of atropine, it is long enough to cause serious inconvenience. Duboisin, moreover, has the further disadvantage that it occasionally causes vertigo and even delirium; hence these drugs are seldom used except when atropine, as occasionally happens, causes conjunctival irritation.

A much more useful agent is the hydrobromate of homatropine, as its effect entirely passes off in from twenty-four to thirty-six hours. Whether it can be relied upon in children, and in cases of spasm of the accommodation, to produce complete paralysis, is a point which more extended experience is needed to determine. It is, however, quite efficient, in ordinary cases, if used of a strength of 1 per cent. (4 gr. to the oz.) and at short intervals. As the effect is so transient, it is probably unnecessary to prescribe its use for several days; the best plan is to let the patient use it three times on the morning of his visit, at intervals of half an hour, and every ten minutes during the hour preceding examination. The combination of hydrochlorate of cocaine 2 per cent. with the homatropine appears to increase its effect both in the dilatation of the pupil and in the paralysis of the ciliary muscle. The first application, however, of this mixture causes considerable smarting, and so is likely to be resented by children at the second instillation.

The effect of a cycloplegic on a hypermetrope is to render his distant vision worse. The eye being under the influence of atropine, the hypermetropia is again tested by convex lenses until the one is found which gives the best result; this should be at least as good as that obtained before the use of atropine.

$$\text{Example.}—\text{Date ; } L V = \frac{6}{9} + 1.5 \text{ D} = \frac{6}{6}$$

$$\text{Date (Atrop^d) ; } L V = \frac{6}{24} \bar{c} + 2.5 \text{ D} = \frac{6}{6}$$

In this case the *manifest* hypermetropia is 1.5 D, and the *total* 2.5 D; the amount of *latent* H. therefore is 1 D.

If vision is improved by convex lenses up to a certain point, but not to the normal standard, *compound hypermetropic astigmatism* (see p. 503) may be suspected to be present. If the result obtained by convex lenses is not as good as that which was obtained before atropine was used, astigmatism is almost certainly present.

B. Convex lenses render vision worse.—(a) *Distant vision is normal.*—The condition cannot be *myopia*, but may be (i) *emmetropia*, or (ii) *latent hypermetropia*.—Latent hypermetropia may be suspected if the patient is under thirty, if the symptoms are those of hypermetropia (see p. 500), and if the near-point is farther away than it should be at the patient's age. The diagnosis can generally be established by the ophthalmoscope (see Retinoscopy). If these symptoms are absent, and if the ophthalmoscope fails to discover any hypermetropia, we may assume that the eye is *emmetropic*. If, however, symptoms are present and persist, it is often advisable to paralyse the accommodation; if distant vision is then still normal, the eye is *emmetropic*; if it is impaired, *hypermetropia* is present, and the amount must be ascertained in the manner already described.

(b) *Distant vision is subnormal.*—The condition is either (i) *myopia* (or spasm of the accommodation), or (ii) *astigmatism*.

(i) *Distant vision is improved by concave spherical lenses.*—Near vision is good—*i.e.* 0.50 Sn. can be read fluently; the near-point is nearer than corresponds with the patient's age, *myopia* is present.

Spasm of the accommodation may cause an eye to resemble myopia in all respects—indeed, an eye under such circumstances is to all intents and purposes myopic for the time; spasm sufficient to produce this condition rarely occurs, however, except in children, and, if the accommodation be paralysed by the use of atropine—and in children it is always safer to do this—the true refraction of the eye can be ascertained.

If concave lenses bring distance up to the normal standard,

the case is one of myopia only. If vision is improved, but not to the normal standard, the case is one of *compound myopic astigmatism*¹—see below, under (ii).

Before endeavouring to find the lens which corrects the myopia, the *far-point* should be ascertained; this is done by finding the smallest of the reading-types which the patient can read (which in uncomplicated myopia is 0.50 Snellen), and then ascertaining what is the greatest distance at which he can read it. For example :

$$V < (\text{less than}) \frac{6}{60}, \text{ \& } 0.50 \text{ Sn. at } 20 \text{ cm. (p.r.)}$$

If the punctum remotum of a myopic eye can be with accuracy determined, the degree of error is manifest; for the

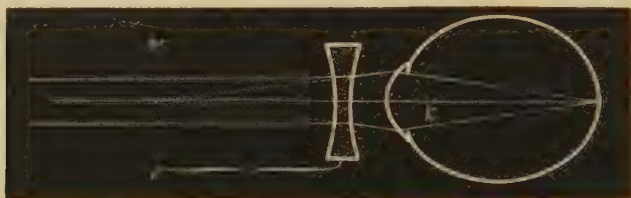


FIG. 146.—Parallel Rays entering a Myopic Eye through a Biconcave Lens.

lens, the focal distance of which corresponds to the distance of the punctum remotum, is the measure and, at the same time, the correction of the myopia present.

The focus of this lens must coincide with the far-point of the eye, so that parallel rays will be rendered as divergent as they would be if they came from the far-point (see fig. 146). They will therefore be focussed on the retina without any accommodation being used, and distant objects will be seen under the same conditions as in emmetropia. A stronger lens will render the rays more divergent; but, as they can still be focussed on the retina if the accommodation is called into play, vision is not necessarily rendered worse. Hence the *weakest* concave lens which gives the best vision is the glass to be chosen.

¹ Assuming, of course, that the defective vision is due to an error of refraction; it must be remembered, however, that morbid changes in the fundus are very frequently met with in *myopia*.

Example.—

$$L V < \frac{6}{60}, \text{ \& 0.50 Sn. at 20 cm. (p.r.) } \bar{c} = 5 \text{ D, } V = \frac{6}{6}.$$

A lens with a focal length of 20 cm., or one-fifth of a metre, is one of 5 D (see p. 507).

In *high degrees* of myopia, however, the 'far-point' cannot be accurately determined in this manner. For, owing both to the nearness of the type and the length of the eye, the retinal images are so large that their form can be recognised, even when they are not accurately focussed. Hence the letters of 0.50 Sn. can sometimes be read, even when placed *beyond* the patient's far-point. On the other hand, if, as is frequently the case, there are morbid changes in the fundus, which have lowered the visual acuity, the patient may find it necessary to hold the type at a *shorter distance* than the true 'far-point' of the eye.

If a concave lens improves vision, but does not bring it up to normal standard, and there are no other morbid conditions, the case is one of *compound myopic astigmatism*.

(ii) *Distant vision is not improved by concave spherical lenses.*—The case is probably one of *simple* or *mixed astigmatism*. Astigmatism is also present when the distant vision cannot be brought up to the normal standard by spherical lenses [see above, under (i)].

Subjective Tests for Astigmatism.—(a) *If vision is improved by spherical lenses, compound hypermetropic astigmatism or compound myopic astigmatism* is present. Place the lowest sphere which gives the best distant vision in front of the eye in an astigmatic trial frame (fig. 138)—(always test one eye at a time, keeping its fellow covered); in front of this place a stenopaïc slit (a black metal disc with a slit running across its centre, 25 mm. long and 1 mm. wide), and rotate it slowly until the best distant vision is obtained. Try now if a lower sphere improves the vision—if so, substitute it. The sphere with which the best vision is obtained is the measure of the refraction in this, the meridian of least error. Now, with this correction, turn the disc round at right angles to its present position so that the slit is parallel to the meridian of greatest

error—the test-types become indistinct again. The sphere which now gives the greatest improvement in vision is, in combination with the weaker sphere in the frame, the measure of the error in this, the greater ametropic meridian. Change this last sphere for a cylinder lens of equal strength, fixing it in the trial frame in the place of the stenopaïc slit with its axis at right angles to the position the slit last occupied. This combination of lenses is the correction of the astigmatic ametropia.

(b) *Vision is not improved by spherical lenses.*—Simple hypermetropic astigmatism, simple myopic astigmatism, or mixed astigmatism is present. Place a stenopaïc slit in front of the eye and rotate it slowly; if in any meridian vision is normal, turn the slit at right angles to it, *i.e.* parallel to the ametropic meridian, and try spherical glasses; the sphere which now gives normal vision is the measure of the ametropia in this meridian. In its stead place a cylinder with its axis at right angles to the direction the slit occupied last—the error will be corrected by this glass.

If vision cannot in any meridian be brought to normal by the use of the stenopaïc slit, *mixed astigmatism* is present. Place a low sphere, convex or concave (+1 D or -1 D), behind the slit, and turn the latter slowly round until the best meridian is found. Try if a strong sphere can be borne; if so, substitute the one which gives best distant vision. Now rotate the disc till its slit is at right angles to this meridian, and try spherical glasses of the opposite kind to that first used; the sphere which gives the best distant vision is, with the strength of the first sphere subtracted, the measure of the error in this meridian. It must of necessity be a stronger sphere of the opposite kind to the one first used, in order, first of all, to neutralise it and then to exert its required power. A cylinder of equal strength substituted for the slit with its axis at right angles to the position which the latter occupied will be the required correction.

In testing with the stenopaïc slit, the results are as a rule unsatisfactory unless it is known that the accommodation is in abeyance. Individuals under thirty years of age, therefore, should be atropinised, otherwise the above tests are unreliable.

Example 1.—

\$=stenopaïc slit with slit vertically placed. S=the same with slit horizontal. V=vision. <=less than. ⊃=combined with.

$$R \ V < \frac{6}{60} \ \bar{c} + 2.5 \text{ D sph.} = \frac{6}{18} \text{ not further improved by spheres.}$$

$$\bar{c} + 2.5 \text{ D sph.} \supset \$ = \frac{6}{9} \text{ (now try a lower sphere).}$$

$$\bar{c} + 2 \text{ D sph.} \supset \$ = \frac{6}{6}.$$

$$\bar{c} + 2 \text{ D sph.} \supset S = \frac{6}{36}; \text{ i.e. worse.}$$

$$\bar{c} + 2 \text{ D sph.} \supset S \text{ and } \supset + 3 \text{ D sph.} = \frac{6}{6};$$

$$\text{i.e. } R \ V < \frac{6}{60} \ \bar{c} + 2 \text{ D sph.} \supset + 3 \text{ D cyl., axis vertical} = \frac{6}{6}.$$

(Compound Hypermetropic Astigmatism.)

Example 2.—

$$L \ V < \frac{6}{60} \text{ not improved by spheres.}$$

In no meridian can the slit give perfect distant vision.

$$L \ V = \frac{6}{36} \ \bar{c} - 1 \text{ D sph.} \supset \$ = \frac{6}{12} \text{ not improved by rotation of slit;}$$

$$\bar{c} - 2 \text{ D sph.} \supset \$ = \frac{6}{6}.$$

$$\bar{c} - 2 \text{ D sph.} \supset S = \frac{6}{36}.$$

$$\bar{c} - 2 \text{ D sph.} \supset S \text{ and } \supset + 3 \text{ D sph.} = \frac{6}{9}.$$

$$,, \quad ,, \quad ,, \quad + 4 \text{ D sph.} = \frac{6}{6};$$

$$\text{i.e. } L \ V = \frac{6}{36} \ \bar{c} - 2 \text{ D sph.} \supset + 4 \text{ D cyl., axis vertical} = \frac{6}{6}.$$

(Mixed Astigmatism.)

A better combination would be + 2 D sph. \ominus - 4 D cyl., axis horizontal; for, wherever it is possible, the axis of the cylinder should be placed horizontally to avoid any prismatic effect that would occur if its refracting meridian be placed in that, the horizontal meridian.

Another subjective test for astigmatism with the stenopaïc slit is by the use of radial lines, and depends upon the recognition of lines instead of letters; the vision, therefore, cannot be estimated without also referring to Snellen's distant test-types.

Lines about a foot in length radiating from a common centre, with angular separations of 30° , should be placed at a distance of 6 m. in a good light; one line will appear black and distinct if the meridian at right angles to it be emmetropic, or has been rendered so by a correcting sphere; in all the other meridians they will appear blurred and indistinct, especially those in the emmetropic meridian (see p. 504). The meridian with the distinct lines and the meridian at right angles to it are the principal meridians.

Place a stenopaïc slit in front of the eye and rotate it until all the lines are seen with equal clearness and definition: this is the emmetropic (or corrected) meridian. Rotate the slit at right angles to this meridian parallel to the ametropic meridian: only the lines which were seen clearly prior to the use of the slit are now seen distinctly, just as if the slit had been removed. The sphere which now renders the lines in all meridians equally distinct is the correcting lens for this meridian, and a cylinder lens should be substituted with its axis at right angles to this, the ametropic meridian.

ii. **Other Subjective Tests.**—There are other subjective tests for astigmatism, some of which it will be well to mention briefly. Various instruments, called *optometers*, have been devised for the purpose of facilitating the estimation of the refraction; most of these consist essentially in an arrangement, more or less ingenious, by which lenses of various powers and in different combinations can be rapidly placed in succession in front of the eye which is to be examined, the test-object being Snellen's types; these require no further description here.

An attempt was made a few years ago to utilise the prin-

ciple of a very old test known as *Scheiner's*. This depends on the fact that an emmetropic eye, looking at a point of light through two minute apertures, placed close together, sees the point singly, because the rays which pass through each aperture meet at the same point on the retina; but an ametropic eye under the same circumstances sees the point *reduplicated*, because the rays coming through the two apertures would meet in a single point in front of the retina in myopia, and behind it in hypermetropia, and would therefore, in either case, cut the retina in two separate places; the test, however, is not sufficiently accurate for practical use.

Thompson's ametrometer is an ingenious instrument, but labours under the same disadvantage as the preceding test. In it two small flames are looked at from a distance, and the size of the projected images of the circles of diffusion which they form on the retina is measured by finding the extent to which the flames must be separated in order that their images should appear to touch each other, but not to overlap. The two lights can be placed in any meridian, and the calculation made for the meridian of the eye which is at right angles to it.

Among *objective tests*, the use of the ophthalmoscope takes the foremost place, and it may be employed in several ways; but they have this feature in common, that the result depends on the direction given by the refracting media of the eye to the rays which are reflected from the retina, these rays being, as we have seen, *parallel* in emmetropia, *convergent* in myopia, and *divergent* in hypermetropia.

iii. **Testing by Direct Ophthalmoscopic Examination.**—This test was very warmly advocated by Couper some years ago, when objective tests were little used, and has been extensively practised by him and others; as an approximate test it is exceedingly useful, and its employment should be cultivated by every one. To render it an accurate test requires considerable experience, and even with this it is in most hands inferior to some other methods, at any rate in astigmatism.

An ophthalmoscope containing a series of convex and concave lenses is necessary, and the surgeon must be able to relax his own accommodation. We have seen that in emmetropia the rays from the fundus are parallel on leaving the eye, and

that therefore they are focussed on the surgeon's retina (if his accommodation is relaxed) when his eye is placed close behind the sight-hole of the mirror, and when the latter is held close to the eye under examination. If the rays coming from the eye are parallel, as in emmetropia, a convex lens will render them convergent, and they can then be no longer focussed by the observer's eye. Hence—

In emmetropia the fundus is clearly seen without any lens behind the mirror, and a convex glass renders the image blurred.

In *myopia* the rays are *convergent* on leaving the eye; hence the fundus cannot be seen by an emmetropic eye until they have been rendered parallel by a concave lens. The weakest concave lens which makes the details of the fundus clear is the measure of the *myopia*—provided of course that neither the patient nor surgeon is using his accommodation; if a stronger lens is used, the rays are rendered divergent, and can then be focussed only by the surgeon using his accommodation; this, however, is done instinctively, and by most people unconsciously, so that the fundus is still clearly seen with a concave lens much stronger than that required to correct the *myopia*. It follows from what has been said that—

In myopia the fundus can be seen only by using a concave lens; and the weakest concave glass with which it can be seen is the measure of the myopia.

In *hypermetropia*, the patient's accommodation being relaxed, the rays leave the eye *diverging*; a clear view of the fundus can therefore be obtained only by the surgeon using his accommodation. Most people are unconscious of the act of accommodation, and therefore, seeing the fundus clearly, may think that the eye is emmetropic; but if a convex glass be now placed behind the mirror, the accommodation partially relaxes, and the fundus is still clearly seen. It follows therefore that—

The fundus of a hypermetropic eye can be seen with a convex lens; and the strongest convex lens with which it can be clearly seen is the measure of the hypermetropia.

To recapitulate.—The patient's accommodation being relaxed.—If the fundus is clearly seen *without any lens* behind

Plate XVII.

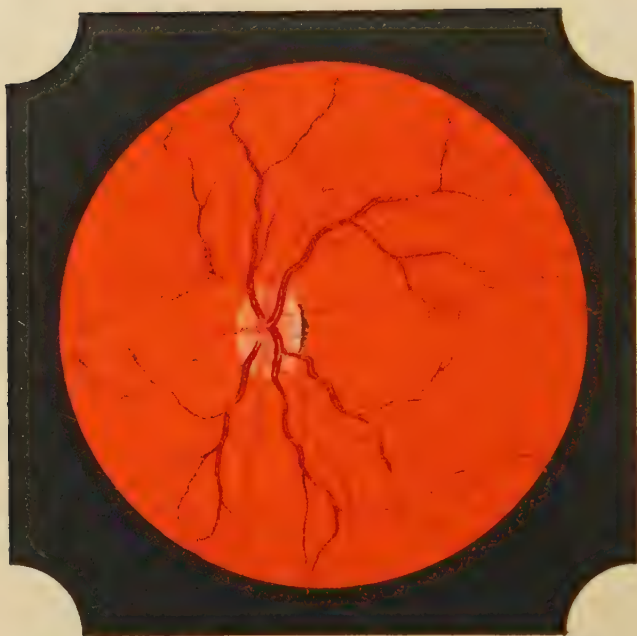


Fig. 1.—Astigmatism (*Vertical vessels in focus*).

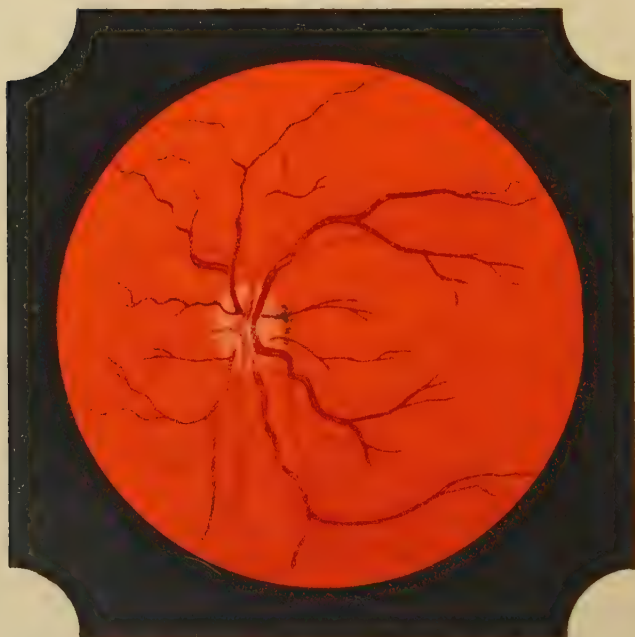


Fig. 2. The same fundus as above (*Horizontal vessels in focus*).

the mirror, the case may be one of *emmetropia* or *hypermetropia*, but cannot be myopia. If a convex lens renders the image blurred it is *emmetropia*, if it remains distinct it is *hypermetropia*. If the fundus can be clearly seen *only with a concave lens it is myopia*.

The weakest concave lens is the measure of the myopia.

The strongest convex lens is the measure of the hypermetropia.

In *astigmatism* the disc appears to be of an oval shape, and the vessels which run in different directions are viewed under different conditions; thus, supposing the eye to be emmetropic in the horizontal meridian, and myopic in the vertical, the vertical vessels will be clearly seen, but the horizontal will require a concave lens to render them distinct. This follows from what was said on p. 504, for since the rays from any point on the retina which come out through the horizontal meridian are parallel, they are focussed on the observer's retina, and by the rule there given a vertical linear image of the point will be formed on the observer's retina; if now a vertical vessel be looked at, it is seen clearly, because all its points form elongated vertical images which overlap one another; the horizontal vessel, on the contrary, looks blurred, because the images of its points are elongated, not in the direction of its length, but across it; but if a concave lens is placed behind the mirror, of such a strength as to bring the rays coming through the vertical (myopic) meridian to a focus on the retina, the horizontal vessels will be clearly seen (see figs. 1 and 2, on opposite page).

Hence we get this rule for the estimation of astigmatism:

The refraction of either principal meridian can be ascertained by finding the weakest concave or strongest convex lens with which the vessels whose course is at right angles to that meridian can be seen.

With practice it is possible to estimate astigmatism with great accuracy by this method in most cases, but there are several difficulties. On the disc itself vessels can usually be found running in several directions, but the refraction of the region of the *yellow spot*, and not that of the optic disc, is what is required, and in the region of the macula there are but

few vessels to be found, and these frequently do not lie in the principal meridians. Tempest Anderson has endeavoured to remove this difficulty by an ingenious apparatus by which an image of fine wires radiating from a common centre is thrown on the retina, those of the lines which correspond to the principal meridians of refraction being used as test-objects.

iv. Testing by the Indirect Method of Ophthalmoscopic Examination.

Upon examining the fundus by the indirect method, if the details appear smaller than natural, myopia may be suspected; if larger, then hypermetropia is present.

In emmetropia the image of the disc remains of the same size, whatever is the distance of the lens from the observed eye.

In myopia the image of the disc enlarges as the lens is withdrawn from the eye.

In hypermetropia it diminishes.

In astigmatism the shape of the disc appears to change as the lens is withdrawn; when the latter is close to the eye, the disc appears oval, the long diameter corresponding to the meridian of least refraction (which is the reverse of what occurs in the direct method); as the lens is withdrawn the relative size of the diameters changes until the long axis corresponds with the meridian of greatest refraction.

v. Testing with Mirror alone, held at a distance.—

(a) *Fundus-image test.* (b) *Retinoscopy.*—In addition to the methods of using the ophthalmoscope already described, the two following are very useful in estimating refraction; in both the ophthalmoscopic mirror alone is employed, and is held at a considerable distance from the eye. The first of these may be called the ‘fundus-image’ test; the other has been called ‘retinoscopy,’ but would be more appropriately designated by some such term as ‘shadow-test.’

(a) **Fundus-image Test.**—If the mirror be held at a considerable distance from an *emmetropic* eye, no image of any details of the fundus is seen, but only a red reflex; this is because only the very minute point of the fundus is seen, which lies on the axis along which light is reflected into the eye, for the rays from any other point on the fundus form a pencil of rays parallel to the axis on which the point is situated, so that by

the time the rays from any two such points have reached the distance at which the observer's eye is placed the two pencils are widely separated (fig. 147).

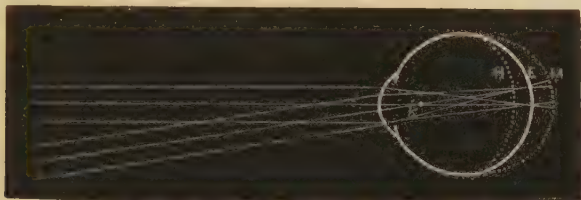


FIG. 147.—Emergent Rays in Emmetropia.

In myopia and hypermetropia, however, a portion of the fundus is seen whose extent is in proportion to the degree of

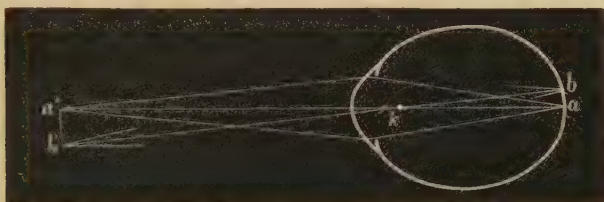


FIG. 148.—Emergent Rays in Myopia.

ametropia. In *myopia*, as we have seen, a real inverted image ($a' b'$, fig. 148) of the fundus is formed in the air at the patient's

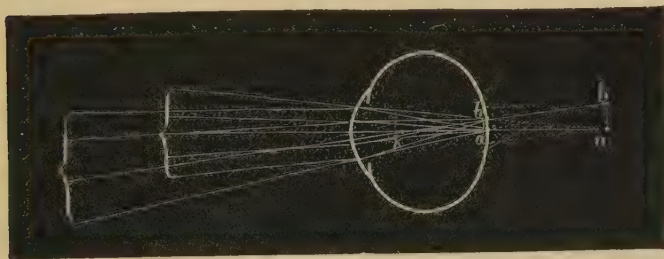


FIG. 149.—Emergent Rays in Hypermetropia.

'far-point,' and, since this is in front of the patient's eye, it appears when the observer's head is carried from side to side to move in the *opposite* direction.

In *hypermetropia* the emerging rays form divergent pencils; hence the image of the fundus is virtual and erect, and is formed behind the patient's eye (see fig. 149); it therefore appears to move in the *same* direction as the observer's head.

Hence we get this rule:

If, while the mirror is held two feet or more from the eye, any details of the fundus are seen, the eye is either hypermetropic or myopic. If the vessels move in the same direction as the head, it is hypermetropic; if in the opposite direction, it is myopic.

(b) **Retinoscopy, or the Shadow-test.**—When light is reflected into an eye from a mirror held at a distance of a little over a metre, and the mirror rotated to and fro on one of its axes, the appearance seen through the sight-hole of the mirror varies with the refraction of the eye.

Let the person under examination sit in a dark room with the light above and slightly behind the plane of the head. The examiner should sit in front at a distance of one metre, and reflect the light with the larger mirror of the ophthalmoscope into the eye to be tested.

In one position of the mirror the whole pupil is occupied by a red reflex, but if it be rotated slightly this red reflex shifts its position on the fundus, so that a limited area of the pupil becomes darkened, and the appearance presented is that of a shadow creeping a short distance over the pupillary area (fig. 150).



FIG. 150.

This darkened portion of the pupillary reflex is due to the shadow of the iris on the fundus.

If a *concave mirror* be used the 'shadow' appears to move in the *same* direction as the rotation in myopia, and in the *opposite* direction in hypermetropia.

If a *plane mirror* be used this phenomenon is reversed: the 'shadow' appears to move in the *opposite* direction in myopia, and in the *same* direction in hypermetropia.

Before considering how these facts may be utilised as an accurate test, not only of the kind of error, but also of its degree, it would be well to explain the *rationale* of the phenomena.

When rays of light from a lamp (L, fig. 151) fall on a concave mirror (M_1) they are rendered convergent, and an *inverted* image of the lamp flame (l_1) is formed in the air just beyond the principal focus of the mirror, which should be about 25 cm. From this aerial image the fundus is illuminated. If the mirror be rotated in any direction—say downwards—as to M_2 the aerial image of the lamp flame will move in the same direction; as to l_2 , see fig. 151. If the eye is only slightly myopic, so that its 'far-point' coincides with the position of l_1 or l_2 , a well-defined *erect* image of one of these will be formed on the retina at l'_1 or l'_2 ; and since the

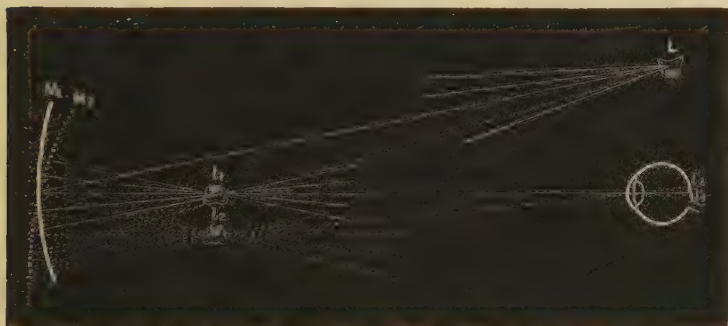


FIG. 151.—Positions of Lamp, Mirror, and Eye in the Shadow-test.

relative position of external objects is inverted on the retina, the lower the aerial image the higher will be that on the retina.

But since it is exceptional for the far-point of a myopic eye and the aerial image of the lamp to coincide, the usual illumination on the fundus is an *erect diffusion-image* of the *inverted* aerial image of the lamp.

This diffusion-image or *fundus-illumination*, taking the shape of the pupil, is usually circular in outline, but may be oval should astigmatism exist.

Its size may vary in two ways:

- (a) *According to the size of the pupil.*
- (b) *According to the amount of refractive error.*

The pupil is usually dilated, and may be considered a fixed

quantity ; but, with regard to the latter, it may vary considerably, thus :

A. The greater the error—

1. *The larger the diffusion-image ;*

therefore : (a) the feebler the illumination ;

(b) the less definite the shadow (from diminished contrast).

2. *The nearer the image (real or virtual) of the fundus-illumination to the nodal point of the dioptric system ;*

therefore : (c) the slower the movement of the shadow ;

(d) the shorter the distance it moves over the illuminated pupil.

B. The less the error—

(a) the brighter the illumination ;

(b) the more definite the shadow ;

(c) the more rapidly it moves ;

(d) the larger the area of the pupillary reflex it travels over.

It is evident that the shadow will move more slowly the nearer the image of the fundus-illumination is to the nodal point of the eye, because it travels through an arc of a smaller circle.

The fundus-illumination always moves in an opposite direction to the movement of the reflected image of the lamp, whatever the error of refraction and whatever kind of mirror (plane or concave) is in use.

This fact is readily understood by referring to figs. 153 and 155. If a concave mirror is used, the fundus-illumination moves upwards upon downward rotation of the mirror, because the inverted aerial image of the lamp in front of the mirror moves downwards. (Sect. I. A. p. 451.)

If a plane mirror is used, the fundus-illumination moves downwards upon downward rotation of the mirror, because the virtual erect image of the lamp behind the mirror moves upwards. (Sect. I. A. p. 447.)

In taking the refractive errors *seriatim*, a concave mirror will be considered in use.

It is well to thoroughly understand that it is the move-

ment of the *image* of the fundus-illumination, not the fundus-illumination itself, which demonstrates the kind of ametropia.

In **Hypermetropia** (fig. 153), a *virtual erect image* $k' l'$ of the fundus-illumination $k l$ is formed behind the eye. If the mirror is rotated downwards, the inverted aerial image $L K$ of the lamp moves downwards in the direction of the arrow; consequently the virtual erect image $k' l'$ moves upwards, the shadow of the iris appearing below and moving in an upward direction, *i.e.* in an opposite direction to the rotation of the mirror (figs. 152; 153).



FIG. 152.

In **Myopia** (fig. 155), on the other hand, a *real inverted image* of the fundus-illumination is formed in front of the eye at its far-point.

If the mirror is rotated downwards, the inverted aerial image of the lamp moves downwards; and, since the fundus-

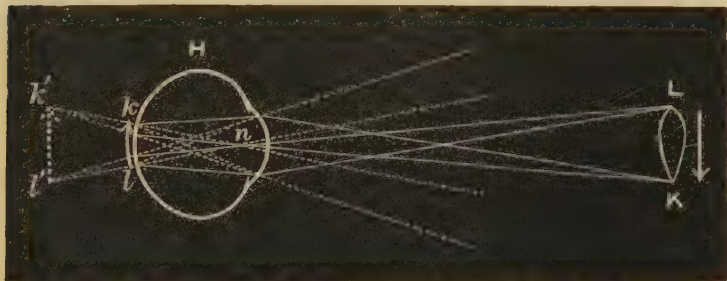


FIG. 153.—Shadow-test in Hypermetropia.

illumination moves upwards, its inverted image, situated at the *p.r.* of the myopic eye, will move downwards; consequently the shadow of the iris will appear to move in the same direction as the rotation of the mirror (see figs. 154, 155).

In fig. 155, $L K$ is the inverted aerial image of the lamp, and M a myopic eye. $L K$ forms an erect diffusion-image $k l$ on the fundus. Rays coming from any point of this diffusion-image will come to a focus at the *p.r.* of the eye and form an inverted image $l' k'$. If $L K$ moves downwards, $k l$ will move upwards, therefore its image $l' k'$ will move downwards; since



FIG. 154.

$l' k'$ is the image beheld by the observer, the shadow will also be seen moving from above downwards; should, however, the mirror be situated at the far-point, *i.e.* at the focus of the emergent rays from the fundus-illumination, no definite shadow will be seen. If, therefore, the observer be seated at the distance of one metre from the patient, and see no shadow upon rotation of the mirror, he may take it for granted that the patient has one dioptré of myopia, since the observer will be situated at his *punctum remotum*.

Again, should the far-point be situated beyond the mirror, behind the head of the observer, the emergent rays would be so slightly convergent that for practical purposes they may

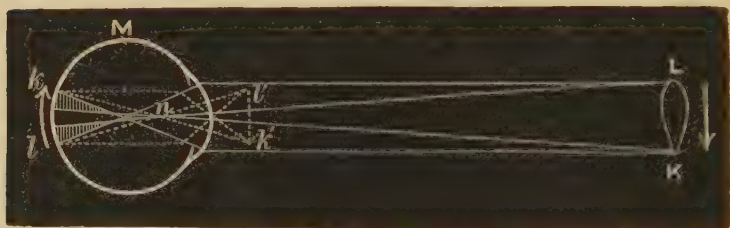


FIG. 155.—Shadow-test in Myopia.

be considered parallel; consequently the shadow will move in an opposite direction to the rotation of the mirror, as about to be explained in emmetropia.

In **Emmetropia** a shadow is seen which moves in an opposite direction to the rotation of the mirror, similar to a hypermetropic shadow. This is due, as in slight myopia, to the fact that the emerging rays are parallel, or practically so, and therefore do not come to a focus anywhere, unless it be on the fundus of the observer; and so he sees, not a virtual erect image behind the eye as in hypermetropia, nor a real inverted image in front of the eye as in myopia, but the fundus-illumination itself. This illumination moves in an opposite direction to the rotation of the mirror; consequently the shadow is seen to do the same.

In **Astigmatism**, in which the amount of refraction varies in different meridians, it is evident that the shadow will not present similar features in those of greatest and least refraction.

The differences will be—

- (a) *In rate and extent of movement.*
- (b) *In definition.*
- (c) *In direction.*

It has already been stated that the fundus-illumination is oval in astigmatism, the long axis being parallel to the meridian of greatest error; the reflex seen, however, appears circular or similar in shape to the pupil.

This oval nature of the fundus-illumination explains the reason why differences in the shadow exist in the two chief meridians:

The larger and more rapid and defined the shadow, the less the error.

The smaller and less rapid and defined the shadow, the greater the error.

The direction of the shadow points out the nature of the error in the meridian examined.

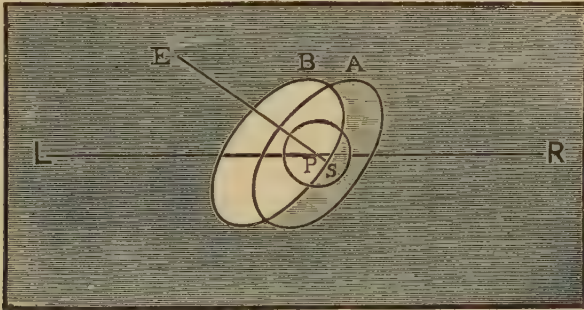


FIG. 156.—The Oblique Shadow in Astigmatism.

If the two chief meridians are obliquely inclined, though the mirror be rotated on its vertical or horizontal axis, the shadows move obliquely.

This phenomenon can be readily appreciated by referring to fig. 156. The circle P is the pupillary reflex, and A represents the fundus-illumination, in an astigmatic eye, in its first position when the whole pupil is illuminated. If the mirror is rotated from left to right on its vertical axis, so that A moves

to B, the shadow s will appear to move upwards, and to the left, in the direction of E.

By retinoscopy, therefore, we arrive at the following conclusions :

1. *If the shadow moves against the rotation of the concave mirror, the refraction is either (a) hypermetropia, (b) emmetropia, or (c) myopia less than 1 D.*

2. *If the shadow moves with the rotation of the concave mirror, the error is myopia more than 1 D.*

3. *If no definite shadow is seen, the error is about 1 D of myopia.*

Again, if the red reflex is dull, the shadow slight, ill-defined, and moves slowly—*the error is great.*

If the red reflex is bright, the shadow marked, well-defined, and moves quickly—*the error is slight or nil.*

The kind and the approximate amount may, thus, be evident upon merely using the mirror.

A plane mirror is equally valuable in retinoscopy, and exactly the same conclusions can be arrived at by using it in a similar manner; the only important difference to be remembered is the reversion of the shadow.

The exact estimate of the degree of error is made by placing lenses of different strengths, convex (+) or concave (—), in front of the eye till the shadow is neutralised, then adding — 1 D to the error discovered.

The practical bearing of what has been said will be made plain by a few examples.

Example 1.—The reflex is dull, the shadow moves in the opposite direction to the mirror, but only a short distance across the pupil; the case is one of high *hypermetropia*.

If convex lenses of increasing strength be now placed before the eye, the reflex becomes brighter, the movement of the shadow greater, but its edge less defined; finally, the shadow becomes indistinct, and then, if still stronger lenses are used, it reappears, moving, however, now in the *same* direction as the mirror, and the eye has been rendered *myopic*.

We have seen that the myopic appearance is produced by the inversion of the fundus-image; and as this occurs at the far-point of the eye, it can only be seen when the latter is

between the patient and observer, and not too near the latter ; for this reason a myopia of 1 D or less cannot be recognised at the distance at which the mirror is usually held, but if the distance be increased a smaller amount can be recognised.

Let it be assumed that the hypermetropia is 8 dioptries in amount : then, with + 6 D, + 7 D, and + 8 D the shadow is still moving opposite to the rotation of the mirror, but it is more distinct and moves more quickly ; with + 10 D the shadow moves with the rotation of the mirror ; this lens, therefore, is more than 1 D over the proper correction ; with + 9 D no shadow is visible, and, since the correction of any error is found by neutralising the shadow and adding - 1 D, then + 9 D \ominus - 1 D = + 8 D, the error of refraction.

Example 2.—The reflex is bright, the shadow moves over a large portion of the pupillary area, and in the opposite direction to the rotation of the mirror—*hypermetropia of low degree, emmetropia, or myopia less than 1 D.* A 1 D convex lens should now be placed before the eye. If the shadow still moves in the opposite direction, *hypermetropia of low degree* is present and must be corrected as in the preceding example. If the shadow disappears, *emmetropia* is present. If the shadow now moves in the same direction as the mirror, *myopia less than 1 D* is present, and a convex lens less than 1 D must be placed before the eye, the strength being determined by the neutralisation of the shadow ; the amount of myopia will be determined by adding - 1 D. Thus, assuming the shadow to be neutralised by + 0.5 D ; then + 0.5 D \ominus - 1 D = - 0.5 D, the error of refraction.

Example 3.—The reflex is very dull, the shadow moving very slightly, and in the same direction as the mirror—*high myopia.* Concave lenses should now be placed in front of the eye, their strength being increased until the shadow is neutralised. A lens a little stronger than this will give the correction. Thus, assuming the shadow to be neutralised by - 12 D ; then, - 12 D \ominus - 1 D = - 13 D, the error of refraction.

Example 4.—The reflex is brighter, the shadow moves in the same direction as the mirror and over a large portion of the pupil—*low myopia.* Correct as in preceding example.

The great advantage of this test, however, consists in the ease with which it can be applied to the estimation of *astigmatism*. The refraction of any one meridian can be ascertained by noting the movement of the shadow in that meridian. This will be made plain by a few examples.

Example 1.—The mirror being rotated on its vertical axis (so that the light moves transversely on the retina), a hypermetropic shadow is seen, neutralised by + 1 D, while on rotating it on its horizontal axis (so that the light moves vertically on the retina) a shadow is seen to move in the *same* direction as the mirror. The case is one of *simple myopic astigmatism*, the horizontal being the emmetropic (an emmetropic eye has a hypermetropic shadow equivalent to 1 D), and the vertical the myopic meridian. In order to correct the error, we use a concave cylindrical lens with its axis horizontal.

Example 2.—We will now suppose that in testing the horizontal meridian (by causing the light to travel transversely) the shadow moves in the *opposite* direction to that of the mirror, and in the vertical meridian there is a similar shadow, which, however, is neutralised by + 1 D, whereas the former requires a much stronger convex lens to neutralise it. The case is one of *simple hypermetropic astigmatism*, and will be corrected by a convex cylinder with its axis vertical.

Example 3.—If in both meridians, after placing + 1 D sph. in front of the eye, the movement of the shadow indicates the same *kind* of error, a difference in its *degree* may be suspected if a difference in the rate of movement is noticed. Spherical lenses must now be placed in front of the eye until one is found which renders one meridian emmetropic. If the meridian at right angles to ~~this~~ still remains ametropic, the case is one of *compound astigmatism* which has been converted by the spherical lens into one of simple astigmatism. Assume that + 2 D sph. neutralises the shadow in the vertical meridian and + 3 D sph. in the horizontal meridian indicated thus,

$$\begin{array}{c} + 2 \text{ D} \\ \text{---} \\ + 3 \text{ D} \end{array}; \text{ i.e. } + 2 \text{ D sph. } \odot + 1 \text{ D cyl., axis vertical,}$$

neutralises the shadows in the chief meridians ;

$\therefore + 1 \text{ D sph. } \odot + 1 \text{ D cyl., axis vertical,}$ is the real error and the required correction.

Example 4.—If, with + 1 D sph. in front of the eye, the shadow moves in the same direction as the mirror in one meridian, and in the opposite direction in the other, the case is one of *mixed astigmatism*. Spherical lenses should now be placed in front of the eye, so as to correct one meridian, thus converting the case into one of simple astigmatism.

For instance—a hypermetropic shadow is seen moving in the horizontal meridian, and a myopic in the vertical; convex lenses are used, and it is found that + 2 D neutralises the shadow in the horizontal meridian. The vertical will, however, have been rendered more myopic, for it was myopic to begin with, and + 2 D has been added. Now neutralise the shadow in the vertical meridian by concave glasses. If - 3 D neutralises the vertical shadow, we write it thus, $-\begin{vmatrix} -3\text{ D} \\ +2\text{ D} \end{vmatrix}$; *i.e.* + 2 D sph. \ominus - 5 D cyl., axis horizontal, neutralises the shadows in all meridians;

\therefore + 1 D sph. \ominus - 5 D cyl., axis horizontal, is the proper correction and the glass required.

If the principal meridians are not exactly horizontal and vertical, but slightly oblique, and the mirror is rotated on its horizontal or vertical axis, the edge of the shadow will coincide, not with the axis of rotation of the mirror, but with the nearest principal meridian, and will therefore indicate the direction of the latter; sometimes, however, it is easier to judge of the direction of the movement than of the exact amount of obliquity of the shadow edge.

We have seen that the *real* movement of the light (and therefore of the 'shadow') on the fundus is along a line at right angles to the axis on which the mirror is rotated. Whatever may be the *real* movement, however, it always *appears* to take place in a direction at right angles to the edge of the shadow. This can be illustrated by the simple experiment of passing a card with its edge held obliquely in a horizontal direction across an aperture (as suggested by Charnley¹); it will then be seen that, although the real movement of the card is horizontal, its apparent direction is along a line at right angles to its edge (see fig. 156).

¹ *Ophth. Hosp. Rep.* X. iii. p. 364.

vi. **Other Objective Tests.**—Among the objective methods of estimating astigmatism must be mentioned the measurement of the curvature of the refracting surfaces of the eye by special instruments.

The *ophthalmometer of Javal and Schiötz* is an instrument for testing the curvature of the cornea, and with it the amount of regular corneal astigmatism can be readily measured.

The cornea of the observed eye acts as a convex mirror, and forms an image of an object of known size situated at a known distance. The size of the image being measured,

we have all the data necessary for calculating the radius of curvature of the cornea, according to the formula on p. 453.

The instrument consists of a pair of porcelain plates (fig. 157, *k l*), which are movable along an arc of a circle *m*, which is graduated so that the distance between the plates can be read

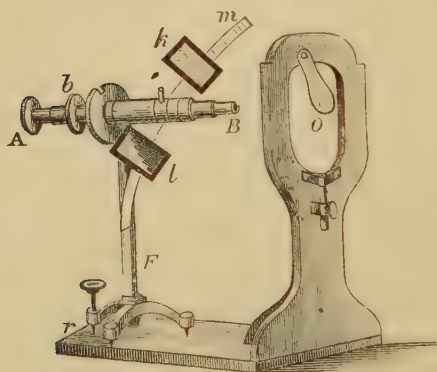


FIG. 157.—Javal's Ophthalmometer.

at a glance. These plates, taken together, constitute the object which is reflected from the cornea of the observed eye at *O*; and the distance between their outer edges is the size of this object.

The image is observed through an eyepiece *A B* containing a double prism of calc-spar, which, being doubly refracting, forms two images of each of the porcelain plates. The two middle images are alone considered, and the construction of the prisms is such that when these two images just touch by their inner edges the size of the image is 6 mm.

The semicircle which carries the porcelain plates can be rotated about an antero-posterior axis, so that any meridian of the cornea can be observed.

It will be noticed that one of the plates (*k*) is rectangular, while the other (*l*) is cut out in six steps. This is for measuring the amount of astigmatism, which is accomplished in this manner: while looking through the eyepiece, turn the semicircle into that meridian in which there is greatest separation of the two central images. This is the meridian of least curvature, since it forms the largest image.

Now approximate the plates until the edges of the central images touch each other (fig. 158).

Next, turn the semicircle through 90°. If there is any astigmatism, the edges of the central images will overlap, and the



FIG. 158.

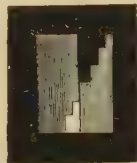


FIG. 159.

number of 'steps' which overlap is the amount of astigmatism in dioptres. Thus fig. 159 shows an astigmatism of 2 dioptres. Irregularity in the shape and sizes of the images points to the existence of irregular astigmatism.

Placido's disc or *keratoscope* is another instrument by which the curvature of the cornea can be examined. It consists of a metal disc about 9 or 10 inches in diameter, supported upon a handle. Upon the disc are painted a number of alternating black and white rings, concentric with a central aperture. The examiner holds the instrument with the aperture close to his eye, and sees the reflection of the rings in the cornea of his patient. If regular astigmatism exists, the rings in the cornea will appear oval, the long axis indicating the meridian of lowest refraction. Irregular astigmatism is easily recognised by the distortion of the rings. The earliest stages of conical cornea are readily discovered by the characteristic corneal reflection.

This test is solely qualitative, and therefore inferior to the preceding. The qualitative nature of the former instrument is also preferable, because the reflection is magnified by a lens in the eyepiece, and smaller errors can therefore be appreciated.

Since astigmatism is more usually due to the state of the corneal curvature than to any lenticular defect, these keratoscopes can be used with considerable success in ophthalmic practice. It must, however, be borne in mind that the lens, through the unequal action of the ciliary muscle, may neutralise

lise the corneal astigmatism ; and unless this be annulled it is obvious that the correction may not produce the expected improvement in vision. The lens, too, may in itself be astigmatic, and may exaggerate corneal astigmatism if it be present.

Section VII.—GENERAL CONSIDERATIONS.

The *use of mydriatics* sometimes entails so much inconvenience on the patient that it is of importance to know in what cases they may be dispensed with.

In myopia the employment of a mydriatic is not as a rule necessary except in the case of young children, or when astigmatism is present.

In patients under twenty, with hypermetropia or astigmatism, the accommodation should as a rule be paralysed.

In those who are slightly older—say, from twenty to thirty—a mydriatic can often be dispensed with, provided that, in the event of the glasses not relieving the symptoms, an opportunity of re-testing can be procured. If a mydriatic is used, homatropine will usually suffice, it being often advisable to wait till its effect passes off before glasses are prescribed.

After the age of thirty mydriatics are seldom necessary.

No hard and fast line can, however, be laid down, and much will depend on individual circumstances. Thus, if a patient is using the eyes for near vision for many hours daily, as is the case with clerks and needlewomen, a very accurate correction is necessary ; if, on the other hand, the eyes are used only for near vision for a short time, an approximate estimation, made without the use of a mydriatic, is sufficient. The fact that a patient has previously worn glasses without relief to the symptoms will also indicate the necessity of a very careful examination.

The treatment of Myopia by glasses.—There has been, and still is, considerable difference of opinion on this subject. The main questions at issue are whether myopes should be ordered the full correction, and whether the subjects of progressive myopia should wear glasses at all.

With regard to both these questions, it may be stated broadly that myopes of whatever degree should wear constantly glasses as near the full correction as possible. Young myopes

of low degree must, as has been already said, be considered possible cases of incipient progressive myopia, and it seems rational to diminish any excess of convergence due to their myopia by the constant use of glasses which render their eyes emmetropic. In these cases the presence of apparent myopia (see p. 495) must be excluded by the use of atropine. If no increase in the myopia takes place within twelve months, the glasses may be used less constantly for reading. This, however, applies only to low degrees, *i.e.* under 3 dioptries. After thirty-five, the glass which corrects the myopia will not suit for near vision, but since a myope of 3 D can read at 33 cm. (13 in.) he does not require glasses for near objects. If, however, such a patient has for several years constantly worn glasses which correct his myopia, he will generally be able to continue using them for all purposes up to the age of forty-five. After this he will require for near vision the addition to his glasses of the presbyopic correction corresponding to his age (see table on p. 478, and p. 549).

If the myopia exceed 3 dioptries, the constant use of spectacles should be insisted upon. At first it may be necessary, even in young adults, to order an under-correction for reading, especially if the myopia is as much as 7 or 8 dioptries; but, as a rule, full or nearly full correction will soon be borne for all purposes, and this condition should be strongly encouraged.

High degrees of myopia are very difficult to treat with glasses. Even young myopes often find it irksome or impossible, when their myopia is corrected, to use the amount of accommodation necessary for near vision; not that this is greater than in emmetropia, but in myopia the structural peculiarities of the ciliary muscle, and the fact that it is seldom or never called into action, render the effort difficult or painful. In all cases it is necessary to exclude the presence of active progressive myopia. The condition does not, however, contra-indicate glasses, but reading and all near work must be strictly prohibited; the lens giving the best distant vision should be ordered, the glass being neutral-tinted. In the absence of all signs of activity, as near full correction as possible should be given for distance, and the strongest con-

cave glass with which the patient can read Snellen 0·5 with comfort and at a suitable distance, for all near work. The danger of under-correcting the myopia is that the patient is liable to look obliquely through the glasses in order to produce a greater effect, but causing thereby an astigmatic condition of the optical apparatus.

In all cases of myopia it is extremely important to determine the total amount of any astigmatism present, and to fully correct this with cylinders.

The treatment of Hypermetropia by glasses.—In ordering glasses for hypermetropia we have to consider whether the patient, having been accustomed to use the accommodation constantly, will be able to completely relax it, as would be necessary for distant vision if full correction were given. A few surgeons do order full correction; but in the case of young subjects it takes a very long time, more patience and perseverance than some possess, for them to get thoroughly accustomed to the glasses; hence it is generally better to deduct something. As to the amount to be subtracted, there is a good deal of difference in opinion and practice. Some correct all the manifest hypermetropia. Others subtract a constant fraction—usually half—of the latent. Others, again, take off a constant amount, as 0·50 or 1·0 D, from the total hypermetropia. A rule which practically works well is to deduct 1 dioptré from the total hypermetropia under the age of twenty, half a dioptré between twenty and thirty, and to order full correction after the age of thirty. If the amount of hypermetropia is above 4 dioptrés, an additional half or one dioptré must be deducted according to the degree and age.

Whether glasses should be worn constantly or only occasionally will depend on the circumstances of each case. Theoretically, no doubt, it is best that the ametropia should be kept constantly corrected, but there are often objections on the part of patients and friends to the constant wearing of glasses. In children with hypermetropia, the constant use of glasses should be insisted on; but in adults with hypermetropia of less than 3 D, it is sufficient if the glasses are worn for near vision. In the higher degrees of hypermetropia, and in astigmatism of 1 D or more, they should be worn constantly.

The treatment of Presbyopia by glasses.—Until the age of forty-five the glass which corrects the eye for distance should theoretically suffice for near vision, and this is actually the case except in myopia of high degree, or myopia which is not corrected until after the age of thirty. After forty-five, however, the natural decay of the function of accommodation (presbyopia) removes the near-point to an inconvenient distance, and the accommodation has therefore to be supplemented by artificial means. The method of ascertaining the presbyopic correction for the emmetropic eye proper to each age is given on p. 481. In ametropia, as a rule, the glass required for near vision is the presbyopic correction, corresponding to the age of the patient, added to the glass which corrects his ametropia.

Examples.—1. A patient aged fifty is hypermetropic to the extent of 1·5 D. The presbyopic correction for the age of fifty is 2 D; he will therefore require for reading $+1\cdot5 + 2 = +3\cdot5$ D.

2. A patient aged fifty-five is myopic to the extent of 1 D. The presbyopic correction is 3 D; therefore he will require for near vision $-1 + 3 = +2$ D.

So that in testing a patient for presbyopia it should first be ascertained whether there is any ametropia, and its amount, and the presbyopic correction then added to this.

There are a few practical points with reference to spectacles which should be attended to. It is essential that they should not only be of the proper strength, but that they should be so fitted that each eye looks through the centre of the glass. So that in prescribing spectacles, when the patient cannot visit the optician, it is necessary to give the distance from the centre of one pupil to that of the other, and to state whether they are to be worn for distance or for reading, &c. Patients often ask whether they should get ‘pebbles’ or glass. There is not much practical advantage in the former, and they are much more expensive; they are lighter and cooler, and may therefore be ordered when the glasses would be of inconvenient weight. In all but the most expensive pebbles, however, the crystal is cut in the wrong direction; and although there may be no flaw visible to the naked eye, such lenses are inferior to glass.

CHAPTER XV.

THE OCULAR MUSCLES.

ANATOMY AND PHYSIOLOGY OF THE EXTRA-OCULAR MUSCLES—BINOCULAR VISION—STRABISMUS—TESTS FOR DIPLOPIA—APPARENT STRABISMUS—PARALYTIC STRABISMUS—SPASTIC STRABISMUS—CONCOMITANT STRABISMUS—HETEROPHORIA—NYSTAGMUS—THE INTRA-OCULAR MUSCLES—PATHOLOGICAL VARIATIONS IN THE SIZE OF THE PUPIL—CYCLOPLEGIA.

ANATOMY AND PHYSIOLOGY OF THE EXTRA-OCULAR MUSCLES.

Anatomy of the Muscles.—Each eye is acted upon by three pairs of muscles, the four recti and the superior and inferior obliques.

All the recti arise from the apex of the orbit by means of two common tendons. The superior common tendon is attached to the upper and outer margin of the optic foramen, and gives origin to the rectus internus, the rectus superior, and the upper head of the rectus externus. The inferior common tendon, or Zinn's ligament, is attached to a depression on the lower and outer edge of the optic foramen, and gives origin to the rectus internus, the rectus inferior, and the lower head of the rectus externus. Between the two heads of the rectus externus is an archway under which pass the two divisions of the third nerve, the nasal nerve, the fourth nerve, and the ophthalmic veins.

As they pass forwards, the muscular bellies diverge from one another, forming a hollow cone which includes the globe. Having become tendinous, they invaginate the capsule of Tenon, and are inserted into the sclera at varying distances from the sclero-corneal junction. Thus the insertion of the rectus internus is 5·5 mm., that of the rectus inferior 6·5 mm., that of the rectus externus 6·9 mm., and that of the rectus superior 7·7 mm. distant from the cornea. The insertions of the rectus superior and rectus inferior are somewhat oblique, the outer fibres being attached more posteriorly

than the inner fibres. The rectus internus has the longest tendon of insertion, the rectus superior the broadest, while the rectus externus has the shortest and narrowest.

Of the four recti, the internus appears to be the strongest, and the superior the weakest.

The *obliquus superior* arises from the apex of the orbit in front of the upper and inner part of the optic foramen, lies along the inner orbital wall above the rectus internus, and reaches the upper and inner part of the orbital margin. Here it passes through the pulley or trochlea, and, under cover of the rectus superior, takes a direction outwards and slightly backwards, to be inserted into the upper and outer part of the globe, as far behind the equator as is the insertion of the rectus superior in front.

The *obliquus inferior* arises by a muscular origin from the inner and anterior part of the floor of the orbit, from a depression on the orbital process of the superior maxilla external to the superior opening of the nasal duct. It passes outwards and slightly backwards between the orbital floor and the rectus inferior, and is inserted under cover of the rectus externus into the external and inferior portion of the posterior part of the globe, near the insertion of the superior oblique but more posterior, at a distance of about 6 mm. from the optic-nerve sheath.

Innervation of the Muscles.—The nerves supplying the muscles of the eye are the third, fourth, and sixth pairs. The *third nerve* (motor oculi) supplies the superior, inferior, and internal rectus, the inferior oblique, the levator palpebræ, the sphincter pupillæ, and the ciliary muscle. The *fourth* (patheticus) supplies the superior oblique. The *sixth* (abducens) supplies the external rectus.

Action of the Muscles.—The muscles of each pair rotate the globe in opposite directions round the same axis; the three axes cut each other in a single point, which remains immovable in all movements, and is therefore called the centre of rotation.

The *centre of rotation* is situated 13·5 mm. behind the cornea, and therefore rather behind the geometric centre of the globe.

The *visual axis* or *line* is the straight line drawn from the yellow spot through the optical centre of the eye; in order that an image may be formed on the yellow spot the object must lie on the visual axis.

The *primary position* is that in which there is a minimum innervation of the ocular muscles; the head is held erect, the

two visual lines are on the same horizontal plane, and are directed straight in front parallel to each other.

The action of any single muscle will be best expressed by the direction in which it causes the centre of the cornea to deviate from the primary position.

The following table shows the direction of the axis of rotation and the action of each individual muscle (see figs. 160 and 161):

Muscles.	Action.	Axis of rotation.
Sup. rectus .	Directs the eye upwards and inwards. Rotates inwards	Horizontal. Inner extremity inclined forwards. Forms an angle of 63° with visual line (fig. 161).
Inf. rectus .	Directs the eye downwards and inwards. Rotates outwards	
Ext. rectus .	Directs the eye outwards	Vertical.
Int. rectus .	Directs the eye inwards	
Sup. oblique .	Directs the eye downwards and outwards. Rotates inwards	Horizontal. Outer extremity inclined forwards. Forms an angle of 39° with visual line (fig. 161).
Inf. oblique .	Directs the eye upwards and outwards. Rotates outwards	

In the above table it is shown that each of the ocular muscles has the power of turning the eye in a definite *direction*; and besides, four of the muscles—superior rectus, inferior rectus, superior oblique, and inferior oblique—have the power of *rotating* the globe on its antero-posterior or visual axis. The recti internus and externus are solely muscles of direction, the remaining four are muscles of direction and rotation.

This rotation will alter the position of the vertical diameter of the cornea by turning its upper end inwards and its lower end outwards, or *vice versa*; and by the change of position of its upper end towards or from the nose, the muscle is said to rotate the eye inwards or outwards.

Associated action of the muscles.—It is evident that if the superior rectus acts in conjunction with the inferior oblique, the inclination inwards caused by the former muscle will be counteracted by the outward movement of the latter; hence a direct movement *upwards* will result. In the same way, if the inferior rectus and superior oblique act together, a *downward* movement is produced. Although a definite action is assigned to each muscle, it must of course be understood that in all the movements of the eyes, as in those of the limbs, all the muscles are concerned, for they are all in a

condition of slight tonic contraction, so that if any one muscle contracts—for example, the external rectus—the other muscles which assist in the outward movement will contract, viz. the obliqui: at the same time a relaxation of the opposing muscles will take place. On the other hand, if a muscle be divided or paralysed, its opponent will cause the eye to deviate; while if they are all divided the globe is rendered perceptibly more prominent.

In fig. 160 the arrows point in the direction of the chief movements of the globe and the muscles which, by acting together, perform them. Those printed in capital letters have

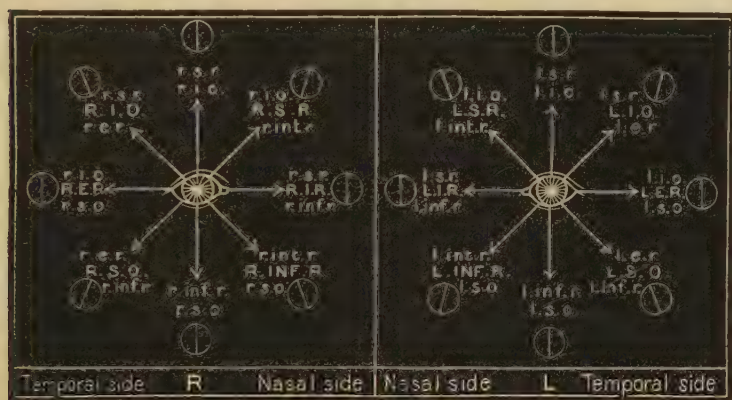


FIG. 160.—Diagram illustrating Ocular Movements.

the power of performing such movements unaided. The normal rotatory movements of the globe in the oblique movements are shown by the diagrammatic outline of the cornea with a line marking its vertical meridian. The muscles which can by themselves perform these oblique movements have individually a greater rotatory power than the combined muscular act; this under normal conditions is in part counteracted by the opposing rotatory action of one of the associated muscles. For example, in looking upwards and outwards three muscles act, the inferior oblique, the superior rectus, and the external rectus: the inferior oblique rotates outwards and the superior rectus rotates inwards; the rotatory action, however, of the inferior oblique is greater in this position of regard than the

superior rectus ; consequently a slight rotation outwards of the globe. In the upward and inward regard the rotatory force of the superior rectus is greater than that of its opponent.

The object is not seen as if it were obliquely placed, as one would expect, owing to the counter-rotation of the head.

Associated movements of the eyes.—All movements of the eyes have for their object the direction of the visual lines to

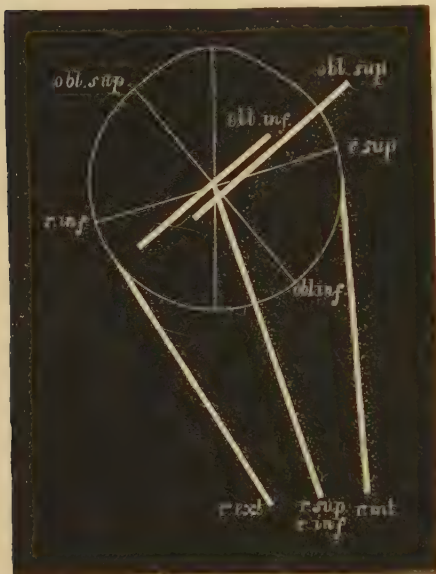


FIG. 161.—Diagram of the Attachments of the Muscles of the Left Eye and of their Axes of Rotation, the former being represented by thick lines, the latter by fine lines. The axis of rotation of the rectus externus and internus, being perpendicular to the plane of the paper, cannot be shown. (After Fick.)

the same point in space ; the movements of the two eyes are therefore necessarily associated. Thus, in looking upwards or downwards both eyes are moved, and the same muscles called into play in each eye. In looking to the right or left both eyes are moved, but the internal rectus of one is associated with the external rectus of the other (*conjugate movements*). Both the internal recti can, however, be called into action and the eyes rotated inwards, so that the visual lines *converge*. It is important to remember that the act of *convergence* is quite independent of

the other conjugate movements ; thus, while convergent, the eyes may be moved upwards, downwards, to the right or to the left, the amount of convergence remaining the same. On the other hand, there may be excessive or deficient convergence without any impairment of the conjugate movements of the globe. Convergence is always associated with contraction of the pupil and the act of accommodation, and in the normal

eye the amount of accommodation used bears a definite relation to the amount of convergence. Thus, looking at a distant object, neither accommodation nor convergence is used, but in proportion as the object is brought nearer, so the greater is the amount both of convergence and accommodation necessary.

Binocular Vision.—When both the visual lines are directed to the same point the image of that point falls upon the yellow spot in each eye, and the two retinal images are combined by the mind to form a single visual impression; this is called *binocular vision*. If, however, while the visual axis of one eye is directed to an object, the other *deviates* from this direction, the condition is spoken of as *strabismus*, or *squint*.

Tests for binocular vision.—It is not always easy to test the presence of binocular single vision. Some tests are too exacting, while others require on the part of the patient more intelligence than is often present. The following are the tests in general use :

i. A prism, base upwards or downwards, is placed before one eye. If diplopia results, binocular vision is probably, but not necessarily, normally present. If diplopia is absent, binocular vision is certainly absent.

ii. If compensatory movement takes place when a prism, base in or out, is placed in front of one eye, with a resulting convergent or divergent strabismus, binocular vision is present.

iii. By means of Hering's drop experiment, the presence of highly developed binocular vision can be determined. The eyes look through a long tube furnished at the other end with a horizontal thread, at the middle of which is placed a bead. The patient is told to fix this object, and to exactly localise falling balls of pith, some of which are made to pass immediately in front of the thread, others behind it.

iv. Snellen's coloured letters (see p. 338) are, on the whole, the best means of determining the presence of binocular vision, since children readily understand what is required.

v. The stereoscope is another excellent means, and suitably chosen pictures make this applicable for children. (See p. 582.)

STRABISMUS.

Strabismus may be defined as *that condition of the eyes in which one of the visual axes deviates from the point of fixation.*

It is evident that in the deviating eye the image of the object on which the other eye is fixed will fall, not on the yellow spot, but on some other part. Thus, if the eye deviates inwards it will fall to the inner side of the yellow spot; if outwards, to its outer side. The mind judges of the position of an object (*projects the image*) by the part of the retina on which the image falls: if on the yellow spot the object is known to be on the visual line; if on the outer side of the yellow spot the object is known to lie to the inner side of the visual line, and so on. Now, in strabismus the mind takes no cognisance of the fact that the eye is deviating, but projects the image as if it were in its true position. Thus, supposing that one eye deviates inwards, the other eye fixes the object; its image, falling on the yellow spot, is projected as lying on the visual axis, and is therefore seen by this eye in its *true* position; but in the squinting eye the image falls to the inner side of the yellow spot, and is therefore projected to a position on the outer side of that which the visual axis would have if the eye were not deviated. Hence two images are seen, a *true* and a *false*, and the displacement of the false image is in the opposite direction to the deviation of the eye.

Thus, in fig. 162, let *r* be the right eye, and *L* the left, which deviates inwards. Let *c* be the centre of rotation, *y* the yellow spot, and *o* the object looked at. The yellow spot in the eye *r* is directed towards the point *o*; that of the eye *L* towards *x*. The image of *o* in the left, or deviating eye, instead of being formed at *y*, the yellow spot, is formed at *o*, and the eye *L*, which judges of the position of exterior objects as if it were in its proper position, projects this image in the direction from which the luminous rays would come, in order that, in a normal position of the eye, the image should be formed at *o*. To find this last direction we have to suppose the eye *L* returned to its normal direction so that the visual line, *y x*, would occupy the position now occupied by *o o*.

Then, since the yellow spot y will be at o , the retinal image o will be displaced inwards at an equal angle and be found at o' . Prolong o' through the centre of rotation to the same distance from the eye as the object, viz. to o' in the visual field; the



FIG. 162.—The Visual Axes in Convergent Strabismus. (Landolt.)

false image will appear at o' , because this is the direction from which the rays would have come if the eye were in its normal position, and the retinal image were at o .

Now, this projection of the object to o' is on the same side as the deviating eye L , and the diplopia is therefore called *homonymous*.

It will be easily seen that, if the eye L had been divergent instead of convergent, the image would have been projected to the opposite side. The anterior part of the eye being thus

turned outwards, the posterior part is turned in the opposite direction, and the image of *o* falls on the outer side of *y*. And since, in the natural state, it is the object situated to the inner side which forms its image on the outer part of the retina, the image is projected in the direction of the nose—that is, to the right of *o*. Under such circumstances the diplopia is said to be *crossed*.

Hence in any case of strabismus where diplopia is present we have the following rule: *The displacement of the false image is always in the direction which is opposite to that of the deviation of the eye.* Thus, when the eye deviates inwards (convergent strabismus), the diplopia is homonymous, not crossed; when outwards (divergent), there is crossed diplopia; when upwards the false image is below; when downwards, it is above.

Tests for Diplopia.—A very simple and ready method of ascertaining the kind of diplopia is to cover the non-deviating eye with a deep red glass by means of a spectacle frame. Then, in a darkened room, we hold a lighted candle about 3 metres in front of the eyes. The patient will then say that he sees two flames, the one red and the other yellow. The red flame is the projection of the image formed upon the eye which has the glass in front of it, the yellow flame belongs to the uncovered eye.

By now interrogating the patient as to the relative positions of the two images, we can ascertain the exact nature of the diplopia. Thus, if the red flame appears on the *same* side as the red glass, the diplopia is homonymous and the deviation is *inwards*; if the red flame is on the opposite side, the deviation is outwards and the diplopia crossed; if above, the deviation is downwards; if below, the deviation is upwards; if downwards and inwards, the deviation is upwards and outwards; and so on for each of the oblique meridians. By this method we are able to detect all degrees of deviation; but it sometimes happens in slight forms of strabismus that the patient can succeed in uniting the double images for some time, and so sees only one flame. Under such circumstances, we have only to place a prism, base upwards or downwards, in front of one eye. This has the effect of separating the two

images vertically, so as to render their fusion impossible; and the patient being unable to correct the vertical diplopia by muscular effort, we can measure the horizontal displacement without difficulty.

Not only the kind but the *degree* of strabismus can be ascertained by this test. *This is directly proportional to the distance between the two images.* It is evident that the distance increases with the degree of the strabismus.

Again, if we direct the patient to follow the light with his eyes, the head being kept at rest, whilst we move the candle in the directions of the various meridians, we find that in paralytic squint the diplopia is increased more in looking in one direction than in another; the distance between the images becomes greater as the eyes are turned in the *direction of the action of the paralysed muscle.*

Again, if we find that during this movement of the eyes in following the flame the distance between the images remains constant, we know that the strabismus is not due to paralysis of an ocular muscle.

Finally, by measuring the actual distance between the images and the distance of the candle from the eye, it is possible to calculate the angle of the strabismus.

Apparent Strabismus.—Before entering into a detailed description of the various forms of squints, it will be advisable to discuss the meaning of the so-called *apparent* or *false strabismus* mentioned above (p. 466), in order that it may be recognised and not be a stumbling-block in arriving at a correct diagnosis. It is a term applied to an apparent convergence or divergence of the eyes which is occasionally observed, but upon careful examination is found to be due to the angle gamma (fig. 115). We are accustomed to judge of the direction of the eyes by the direction of the *optic axes* which pass through the centres of the corneæ; if the angle gamma is large, and the lines of fixation directed towards a distant object, so that they are parallel, the optic axes will then be directed outwards, and so give rise to an apparent divergence. This condition is found only in hypermetropia. Again, if the angle gamma is negative, the optic axes would appear to deviate inwards when the visual axis is directed to

the object of fixation. Such apparent convergence is observed only in myopia. (But see p. 465.)

To distinguish between apparent and real deviation the patient is directed to look steadily at an object held about a metre from the face. If there is no real strabismus, *each* visual line will be directed towards the object, and if either eye is covered, the uncovered eye will still see the object without shifting its position. If, on the contrary, there is strabismus, only *one* visual axis will be fixed on the object, and the other will deviate. If the fixing eye be now covered, the deviating eye must be moved in order to see the object, and by the movement we can judge of the extent and direction of the previous deviation. A still better test is to prove the presence of binocular vision by the use of Snellen's coloured test-types (p. 338).

Three chief divisions of strabismus will now be described, viz. *Paralytic strabismus*, *Spastic strabismus*, and *Concomitant strabismus*.

PARALYTIC STRABISMUS.

Paralytic strabismus is that in which deviation of the visual axis is caused by the paralysis or paresis of one or more of the ocular muscles.

Etiology and Pathology.—The cause of ocular paralysis is usually some lesion of one of the third, fourth, or sixth nerves. It may be *central* or *peripheral*. A central lesion may be *nuclear*, involving the nucleus of origin of one or more of the nerves; *cortical*, implicating their psychomotor centre, which has not yet been localised; or it may destroy the path of connection between these two centres. Direct lesions above the nuclei of origin are so rarely attended with any definite ocular paralysis beyond conjugate deviations to the right or left that they may be practically excluded. A peripheral lesion is one that occurs beyond the nucleus of origin, and may be *intracranial* or *intra-orbital*.

Paralyses from intra-orbital lesions are rare; cellulitis, injury, syphilitic inflammation of the nerve-sheath, or new-growth may be mentioned among the most frequent causes,

and are recognised by their associated local and general symptoms.

Peripheral intracranial lesions are common, and may occur in the substance of the brain (*e.g.* syphilitic gummata) between the superficial and deep origins of the nerves, or in its membranes. The intimate connection of the nerves with the meninges in the vicinity of the cavernous sinus and sphenoidal fissure renders them peculiarly liable to be affected by morbid growth, or in meningitis. Thus, syphilitic inflammation of the dura mater either may result in the formation of gummata which may press on the intracranial nerves, or may form fibrous bands involving the nerve-trunks. Aneurysm of the internal carotid artery in the cavernous sinus, or thrombosis of that sinus, is usually attended with paralysis by direct pressure upon, or inflammation of, the nerves within its walls. Fracture of the base of the skull sometimes involves those nerves either by pressure from the bone, extravasated blood, or by inflammatory exudation. An interpeduncular growth may cause paralysis of both third nerves.

The fourth or sixth nerve is frequently paralysed by increased intracranial pressure caused by a distant lesion, as a new-growth in one or other hemisphere, which as localising symptoms would obviously be of no value.

Nuclear lesions are hæmorrhagic, neoplastic, inflammatory, or degenerative. Hæmorrhages are uncommon in this situation; but, if occurring, may be so extensive as to destroy all the ocular nuclei on either side of the raphé, producing bilateral and complete ophthalmoplegia. Tubercular and syphilitic inflammation, as well as myxoglioma, sarcoma, and other tumours, may, by invading the nuclear region, cause different ocular paralyses. Capillary thrombosis and softening from syphilis, as a result of endarteritis obliterans, by involving these centres, give rise to paralysis of the ocular muscles.

Oculo-motor derangements occur in many spinal and cranial diseases. Disseminated sclerosis is often accompanied by isolated extra-ocular muscular paralyses, some of which are nuclear; there may also be some paresis of convergence. Two forms occur in *tabes dorsalis*—*viz.* recurring and temporary nuclear paralyses in the prodromal stage, and a permanent

peripheral paralysis of one or more muscles (usually the external rectus) in the later stages of the disease. General paralysis of the insane occasionally has, as one of its symptoms, a transitory paralysis owing to involvement of one or more nuclei. Other diseases in which extra-ocular muscles are occasionally paretic are Landry's disease, hydrocephalus, and epidemic cerebro-spinal meningitis. Any increased intracranial pressure may produce unilateral or bilateral internal strabismus, without either sixth nerve being directly involved.

Amongst other causes may be mentioned diphtheria, the external rectus being especially affected; influenza; typhoid fever; rheumatism and cold; poisons, *e.g.* lead and ptomaines; hysteria, though rarely, and always conjugate; and, rarely, Graves's disease. The extra-ocular muscles may be affected in diabetes by what is probably a peripheral neuritis; the third nerve is usually affected. The paralysis may also be congenital.

The muscles most frequently affected separately are the external rectus and the superior oblique. The other recti and the inferior oblique, being supplied by the same nerve, are frequently paralysed together, although separate affections of these are not uncommon.

Ophthalmoplegia, or multiple ocular paralyses, may be partial or complete, unilateral or symmetrical. The cause may be nuclear or peripheral. A progressive nuclear degeneration giving rise eventually to complete paralysis of all the extra-ocular muscles is called by Hutchinson *ophthalmoplegia externa*, in contradistinction to paralysis of all the intra-ocular muscles, *ophthalmoplegia interna*. The latter may follow an attack of diphtheria, but it is more commonly dependent upon progressive central degeneration, and consequently is often associated with the former. Partial ophthalmoplegia may be the result of syphilitic disease of the nerve-centres or of a nuclear hæmorrhage. Again, thrombosis of the cavernous sinus or aneurysm of the internal carotid artery while in that sinus, may cause multiple paralyses; the former is often bilateral and attended with symmetrical symptoms (p. 610).

Associated paralyses.—With the exception of *conjugate deviation of the eyes to the right or left*, associated paralyses are so rarely met with that this form only will be here de-

scribed. It is dependent upon bilateral association of the third and sixth nerve nuclei. The internal rectus of one eye works in harmony with the external rectus of the other eye. The eyes may, from spasm or paralysis, be turned to the right or to the left. These deviations may depend upon an irritative or a destructive lesion which may be nuclear, capsular, or cortical. A *destructive nuclear lesion* causes deviation of the eyes *towards* the paralysed side or *away* from the lesion. Conversely, an *irritative nuclear lesion* would cause the eyes to deviate *away* from the convulsed side, but *towards* the lesion. A capsular or cortical lesion would produce exactly the opposite effect to that of a nuclear. The sixth nerve nucleus of one side being connected with that part of the third nerve nucleus of the opposite side which supplies the internal rectus, a destructive lesion involving the former would cause conjugate deviation of the eyes to the opposite side; and on account of a complete paralysis of the external rectus the eye on the same side as the lesion will not be able to turn *out* as far as the middle line, whereas the opposite eye will be able to turn *in* as far as, but no farther than, the middle line, for the nucleus of the internal rectus muscle is interfered with only through association; it is not destroyed. Since the nuclei of the sixth nerve (abducens) and the seventh nerve (facial) are so closely situated that a lesion of one invariably involves the other, facial paralysis will be present on the same side as the lesion. The sixth nerve nucleus, however, is not the centre for this associated act; the centre is probably in the superior olivary body, destruction of which causes deviation of the eyes to the opposite side without facial paralysis and without complete paralysis of the sixth nerve, both eyes being able to turn as far as the middle line, but no farther. Hæmorrhage into the internal capsule, the common seat of cerebral apoplexy, is nearly always attended with conjugate deviation of the eyes to the same side as the lesion; the head is also turned in that direction. This symptom is not as a rule permanent; it disappears in a few days.

Symptoms common to Ocular Paralysis.—1. There is usually an obvious *squint*. The *primary deviation*, however—that is, the deviation of the affected eye when the healthy eye

fixes—is always less than the *secondary deviation*—that is, the deviation of the good eye when the affected eye fixes. A greater effort than usual is required in order to fix with the paralysed eye; consequently, a greater impulse is sent simultaneously to the two eyes. This strong impulse causes the healthy eye, which responds readily, to deviate considerably.

2. *Diminished mobility* of the affected eye in the direction of the action of the paralysed muscle, and the field of fixation, if tested by means of the perimeter, is found to present a definite limitation according to the muscle affected.

The fields of fixation may be considered as composed of nine divisions. A central division would represent the primary position of the eyes, or the eyes at rest; the remaining eight, secondary positions.

Left upper	Upper median	Right upper
Left lateral	Central	Right lateral
Left lower	Lower median	Right lower

One or more of these divisions would be lost according to the nature of the paralysis.

3. *Diplopia* is generally present. As the eyes are turned in the direction of action of the affected muscle the distance be-

tween the images increases; whereas, if they are moved in the direction of action of the opponent of the affected muscle, the images approach and may coalesce. The diplopia may give rise to *giddiness*, and uncertainty in walking; a tendency to vomiting may be present. These symptoms of vertigo disappear if the sound eye be covered. Short of actual diplopia, there may be an indistinctness of vision.

4. *False projection*.—If the sound eye be covered, and the patient told to touch quickly with the finger an object placed in front of him, he thrusts his finger to one side of it (the side of the paralysed muscle). For instance, supposing the right external rectus to be paralysed, the patient aims too far to the right, for, since his eye is turned inwards to the left, the object appears displaced to the right (see p. 556).

5. *Inclination of the head*.—The head is frequently turned in the direction of the action of the paralysed muscle.

Special symptoms.—*Paralysis of the sixth nerve*.—The *external rectus* is the muscle affected. Here we find that the

eye is deviated towards the sound side and the outward movement of the globe limited. The primary deviation is towards the sound side, while the secondary deviation is towards the diseased side. There is homonymous diplopia: the double images are on the same level in all positions of regard, and in the horizontal plane they are parallel to one another; but upon looking up and out, or down and out, there is an inclination of the false image, in the former movement away from, in the latter towards the true. The affected eye cannot look outwards, so in endeavouring to perform the above movements it turns directly upwards or downwards without any rotation,



FIG. 163.—Paralysis of Right External Rectus.

whereas the sound eye, being able to perform these movements, undergoes the natural rotation; the result is a loss of parallel correspondence of the retinal images, and an inclination of the false image in the visual field (see fig. 163).

The distance between the images increases when the patient looks towards the side of the eye affected, and is usually greater on downward than on upward fixation. Consequently the line of separation between the portion of the field of fixation in which there is single vision and that in which there is diplopia is situated obliquely, its lower end being inclined towards the healthy side. This is due to the fact that on looking downwards there is a greater tendency to convergence.

There is false projection towards the affected side. The patient's face is often turned towards the affected side.

Paralysis of the fourth nerve.—The *superior oblique* is the muscle affected. The movement of the eye is *limited* in the downward and outward direction, and in complete paralysis of this muscle the downward movement is limited also. The primary deviation is upwards and towards the healthy side, whilst the secondary deviation is downwards and towards the



FIG. 164.—Paralysis of Right Superior Oblique.

diseased side. There is homonymous diplopia in the lower part of the field of fixation, the images being superposed.

Owing to torsion of the globe outwards, the image of the affected eye is oblique, its upper extremity being inclined towards the healthy side; it is also the lower of the two, and its obliquity is increased on looking towards the affected side (see fig. 164). The vertical distance between the two images is increased in looking downwards and towards the healthy side. (See p. 572.) The false image generally appears nearer to the patient than the true image. The line of separation between single and double vision is horizontally oblique, its lower extremity being on the affected side. The patient's face is often inclined downwards and towards the affected side. There is false projection downwards and towards the affected side.

Paralysis of the third nerve.—The paralysis of this nerve may be *complete*, or only one or more of its branches may be involved.

Complete paralysis of the third nerve presents a very characteristic appearance. There is slight proptosis, and the upper eyelid falls over the cornea (ptosis).

The pupil is moderately dilated, and does not respond to light. There is paralysis of the accommodation of the affected eye.

The movements of the eye are limited upwards, downwards, towards the sound side, and in intermediate directions. Both the primary and the secondary deviations are outwards, though the primary deviation is sometimes also slightly downwards.

There is crossed diplopia. The false image is oblique, and is inclined towards the affected side; it also appears nearer to the patient and higher than the true image

(see fig. 165). The lateral distance between the images is increased in looking towards the healthy side; the vertical distance increases on looking upwards or downwards; in the former case the false image is above, and in the latter case below, the true image. The obliquity increases in looking towards the diseased side. The patient often inclines his face towards the sound side and somewhat upwards. There is false projection towards the sound side.

There is crossed diplopia. The false image is oblique, and is inclined towards the affected side; it also appears nearer to the patient and higher than the true image

(see fig. 165). The lateral distance between the images is increased in looking towards the healthy side; the vertical distance increases on looking upwards or downwards; in the former case the false image is above, and in the latter case below, the true image. The obliquity increases in looking towards the diseased side. The patient often inclines his face towards the sound side and somewhat upwards. There is false projection towards the sound side.

The lateral distance between the images is increased in looking towards the healthy side; the vertical distance increases on looking upwards or downwards; in the former case the false image is above, and in the latter case below, the true image. The obliquity increases in looking towards the diseased side. The patient often inclines his face towards the sound side and somewhat upwards. There is false projection towards the sound side.

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There is false projection towards the sound side.

Partial paralysis of the third nerve may affect one or more of the muscles supplied by it.

The *internal rectus* is the muscle most frequently involved. Its paralysis is accompanied by external deviation, and by limited movement of the globe inwards. The primary deviation is towards the diseased side, and the secondary deviation towards the healthy side. The diplopia is crossed, the double images being parallel and on the same level (fig. 166). The distance between the images is increased when the patient looks towards the sound side. The line of separation between

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FIG. 165.—R. 3rd N.

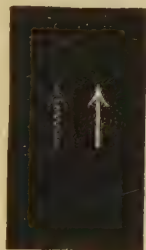


FIG. 166.—R.I.R.

the single and double images in the field of fixation is inclined obliquely, its higher extremity corresponding to the sound side. The patient turns his face in the direction of the affected muscle, *i.e.* towards the healthy side. There is false projection towards the healthy side.

Paralysis of the *superior rectus* is characterised by limited movement of the globe in the direction upwards and slightly towards the healthy side. The primary deviation is downwards, and when the patient looks upwards the affected eye deviates downwards and towards the diseased side. The secondary deviation is upwards and towards the healthy side. The vertical and slightly crossed diplopia increases in looking upwards and towards the diseased side; the obliquity of the



FIG. 167.—R.S.R.



FIG. 168.—R. Inf. R.

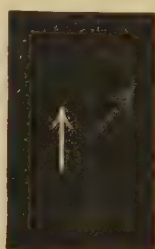


FIG. 169.—R.I.O.

false image increases in looking towards the healthy side (see fig. 167). The patient turns his face upwards and slightly towards the healthy side. There is false projection upwards and towards the healthy side.

The *inferior rectus* is but rarely paralysed alone. When such paralysis exists there is restricted movement of the globe downwards and towards the healthy side. The primary deviation is upwards and towards the diseased side. The secondary deviation is downwards and towards the healthy side. The diplopia is most marked in looking downwards. The images are superposed, they are slightly crossed; the false image is the lower, and its apex is inclined towards the affected side (fig. 168). The vertical separation of the images is increased by looking downwards and to the affected side. The obliquity of the false image increases in looking towards the

healthy side. The patient turns his face downwards and slightly towards the affected side. There is false projection downwards and towards the healthy side.

The *inferior oblique*, when paralysed, is unable to turn the eye upwards and towards the diseased side. The primary deviation is therefore downwards and towards the healthy side; whilst the secondary deviation is upwards and towards the diseased side. The diplopia is most marked in looking upwards when the images are superposed and slightly homonymous. The false image is the higher, and its upper end is inclined outwards (fig. 169). The vertical separation of the images is increased by looking upwards and towards the healthy side; the obliquity increases in looking towards the affected side. The patient directs his face upwards and rather towards the side of the diseased eye. There is false projection upwards and towards the affected side.

Recurrent third nerve paralysis.—This is a definite condition found associated with a definite group of symptoms. Commencing usually in early life, it often persists for many years; the frequency of the attacks varies from ten days to one year. Each attack, as a rule, begins with hemicrania, and the disease has consequently been termed hemicrania ophthalmoplegia. All the branches of the third nerve are almost always involved, and it is very rare for any other nerve to be affected. The disease is never bilateral. The duration of each attack is generally about two days. The exact pathology is unknown. Autopsies of four cases have been recorded in which definite inflammatory changes, or new-growths in or around the nerve-trunk, have been found. Some think a basal lesion is always present; others have suggested a nuclear lesion as the cause. In many cases there is a well-marked hereditary nervous tendency.

The **diagnosis** of ocular paralyses in some cases is very difficult owing to the implication of two or more muscles; on the other hand, a paresis may be so slight that it is hard to discover the eye at fault; secondary deviations may be mistaken for the primary squint in such cases. The difficulty of obtaining clear and decisive answers (for the diagnosis is chiefly made by subjective tests) is so frequent that several

examinations are often necessary before one can arrive at a satisfactory conclusion. The *kind of diplopia* affords the best subjective test.

Figs.¹ 170 and 171 illustrate the relation the false image bears to the true in the visual field. They also demonstrate the fact that the false image is displaced in the direction of the action of the paralysed muscle; and in that position of regard it is well known that there is the greatest vertical separation and the greatest obliquity of the false image.

The true images are represented by thick lines, the false by thin; the dotted line indicates the central line of the figure. The four false images of the right and left superior and inferior recti muscles form, by meeting one another at four points, a diamond-shaped space guarded on either side by a true image; the right true image being to the left, and the left to the right, of the diamond. The four false images of the four oblique muscles by meeting at a point form an **X**, the two true images being represented as a single central image, common to both. In fig. 170 it will be seen that the false images of the right recti muscles are to the left, and the left to the right, of the true, *i.e.* the diplopia is crossed. In fig. 171 the diplopia is seen to be homonymous. Again, the false images are seen to be either above or below the level of the true, and the vertical displacement is greatest in that position of regard indicated by the false image; for example: if, in paralysis of the left inferior oblique, the patient were to try and look upwards and to the left, the false image would be displaced upwards and to the left—that is to say, in the direction of the action of the inferior oblique, the paralysed muscle. This point holds good with all the ocular muscles, viz. that *the false image is displaced in the direction of the action of the paralysed muscle*.

This statement will appear erroneous upon looking at fig. 170, but it must be remembered that in the paralyses of these recti muscles the diplopia is crossed, and so the upper false images incline away from the true; yet, in a paralysis of a right superior rectus, the false image is above and inclined towards the left—that is to say, directed upwards and inwards. Similarly, the false images of the inferior recti are directed

¹ Werner, *Ophth. Review*, vol. v. p. 65.

downwards and inwards. The obliquity of the false images in paralysis of the inferior oblique muscles is away from the central line of the figure and from the true image, i.e. *outwards*; in paralysis of the superior obliques, *inwards*. The same occurs in the rotatory action that those muscles possess upon the vertical meridian of the cornea. This relationship between the obliquity of the false image and the rotatory action of the oblique muscles is the same with regard to the recti muscles. The false images of the superior recti, though above the horizontal plane and inclined from the true images, are, however, inclined towards the middle line, whereas those of



FIG. 170.—Paralysis of the Superior and Inferior Recti Muscles.



FIG. 171.—Paralysis of the Oblique Muscles.

the inferior recti are inclined away. The superior recti rotate the globe inwards; the inferior recti, outwards. (Compare figs. 170 and 171 with fig. 160.)

Differential Diagnosis.—I. Ascertain the absence or presence of any vertical diplopia in any part of the visual fields.

A. If *vertical diplopia is absent*, one of the external or internal recti muscles is at fault.

(1) If the diplopia is *homonymous*, it is one of the external recti.

(2) If the diplopia is *crossed*, it is one of the internal recti.

B. If *vertical diplopia is present*, a superior rectus, inferior rectus, or one of the obliques is at fault.

Ascertain in which quadrant of the visual fields the vertical displacement is at a maximum.

(1) If the displacement is greatest in the right superior

quadrant, either the *right superior rectus* or the *left inferior oblique* is at fault.

(2) If it is greatest in the right inferior quadrant, either the *right inferior rectus* or the *left superior oblique* is at fault.

(3) If it is greatest in the left superior quadrant, either the *left superior rectus* or the *right inferior oblique* is at fault.

(4) If it is greatest in the left inferior quadrant, either the *left inferior rectus* or the *right superior oblique* is at fault.

II. Ascertain which eye is at fault. This is accomplished by finding which is the false image by means of the axiom, *The false image is displaced in the direction of the action of the paralysed muscle.* Where vertical diplopia is present, too much attention must not be paid to any existing lateral diplopia, since a previously existing latent deviation may become manifest and complicate the case, producing, perhaps, an homonymous diplopia with paresis of one of the obliques. The false image often appears to the patient to be nearer him than the true image.

An important point to remember is that a maximum vertical diplopia in the right inferior quadrant points to a possible paresis of the *left superior oblique*, and not the *right superior oblique*. This is due to the fact that though the action of the left superior oblique is to turn the eye downwards and to the left, its downward action is at a maximum when the eye is turned to the right.

After the discovery of the muscle paralysed, it is necessary to localise the seat of the lesion. If only one of the muscles supplied by the third nerve is paralysed, it is, in all probability, a peripheral lesion; it may be nuclear from progressive central degeneration. Nuclear destruction of the sixth is attended with conjugate deviation to the opposite side and facial paralysis of the same side. Gross central can usually be distinguished from peripheral lesions by the presence of severe intracranial symptoms, as persistent headache, vomiting, vertigo, epileptiform seizures, loss of consciousness, the presence of optic neuritis; other paralyses, as facial, anæsthesia of face, hemiplegia, &c.

The majority of ocular paralyses are due to a gummatous infiltration of the nerve-sheaths, associated with pachymeningitis.

The treatment of these paralyses must, as far as possible, be regulated by the cause of the affection. When central disease, of the brain or the medulla, can be traced as the cause of the local affection, the chief malady must be first dealt with. When syphilis is the probable cause, we must have recourse to the iodide of potassium in large doses, with or without the use of mercury. Where the local failure is associated with a rheumatic diathesis, the use of alkalies combined with colchicum, vapour-baths, warm clothing, &c., is advisable. In cases of great debility after acute illness, as diphtheria, typhoid, or other causes, the general health must be improved by the administration of good food, tonics, as ammonia and bark, quinine, iron, and cod-liver oil.

In the use of these therapeutic agents it should be borne in mind that spontaneous recovery from defective muscular action, and even from paralysis, is not unfrequent, also that these cases sometimes fluctuate in their severity from day to day.

Electro-therapeutics are sometimes beneficial here, as in other nerve-lesions, the *primary* (galvanic, continuous) current being employed. The application should be made daily for a period not exceeding ten minutes. One pole of the battery, the cathode, is placed on some distant part of the body (not over the mastoid process, as this is liable to cause vertigo), and the other is placed over the closed eyelids of the affected eye, by means of moist small sponges. The strength of the current should be as great as the patient can tolerate without actual pain. Usually about 3 milliampères is as much as the patient can tolerate. If the conjunctiva be cocainised and a metallic conductor used instead of the sponge, the current can be better localised to the affected muscle by applying it within the palpebral sac near the insertion of the muscle: in this case the current must be weaker, about 1 or 1·5 milliampère.

In addition to these medical and electrical remedies, some precautions may at the same time be taken to prevent, or to alleviate, the discomfort produced by the diplopia. Closure of the affected eye by means of a shade or a disc of ground glass mounted in a spectacle-frame is of the greatest service; it of course prevents the double vision—in fact, the patient generally closes the affected eye of his own accord.

If the good eye be closed, the diplopia certainly disappears, but there is always the false projection of the image in the direction of the action of the paralysed muscle, which produces vertigo, difficulty in judging of the position of surrounding objects, and other disagreeable sensations.

Prisms.—In certain cases which have become stationary, and in which the images are not too widely separated, the employment of prisms proves beneficial in reducing the diplopia, and in stimulating the muscle to renewed action. If we look through a prism we find that it produces an effect similar to that of a pathological deviation; it causes diplopia. It follows, therefore, that by the proper employment of a prism we are often able to neutralise the diplopia. In the use of a prism, the rule is to place its apex in the same direction as that in which the eye deviates: thus, if the eye turns outwards, the apex of the prism must be turned outwards; if the eye turns in, the apex must be inwards also (see p. 455). If there is no deviation, the apex of the prism should be placed in the direction opposite to that of the action of the paralysed muscle. In practice it is well to use a prism slightly below the full correction, so as to give the affected muscle an opportunity of exerting itself; thus, supposing the diplopia to be corrected by a prism of 4° , we should prescribe one of 3° in preference to the former. Another practical point is to divide the prism between the two eyes; thus, supposing No. 6 is found to reduce the diplopia when placed before the affected eye with its apex outwards, we prescribe two No. 3 prisms, one for each eye, and each with its apex outwards. As the impaired muscle regains its strength, the strength of the prism must be diminished.

Operative treatment is never justifiable unless there is evidence of some recuperative power in the paralysed muscle, and all the remedies above mentioned have been duly tried without success. Even after the deviation has become stationary, it is well to wait a few months before resorting to operative measures, inasmuch as spontaneous recoveries sometimes take place in the most unexpected manner. When, however, a muscle has been partly paralysed for upwards of six months, and has resisted all other treatment, an operation

may be of service. This may consist of simple tenotomy of the antagonistic muscle so as to weaken its action, or it may require advancement of the affected muscle. The mode of procedure in these operations is exactly similar to that for concomitant strabismus.

SPASTIC STRABISMUS.

In paralytic strabismus it was seen that the deviation of the visual axis was due to a defective action of one or more of the ocular muscles; in spasmodic or spastic strabismus excessive action is the cause of the squint. These spasmodic affections are extremely rare, but occur sufficiently often to require mention. They may be clonic or tonic, and are usually the result of an irritative lesion which may be direct or reflex. Clonic spasms are not unfrequently seen in the first or irritative stage of tubercular meningitis; rarely, if ever, seen in basic pachymeningitis. Reflex clonic spasms have been described as occurring in children suffering from worms, teething, &c.; they undoubtedly occur, however, in the epileptiform convulsions which result from digestive derangements. Tonic spasmodic action has been observed in epilepsy.

Hysterical strabismus is by no means unknown, and is probably due to excessive rather than deficient action of an ocular muscle; it is usually convergent.

The *treatment* consists in removing all possible causes of reflex irritation, and examining the refraction to avoid mistaking a concomitant strabismus for this affection. Moral treatment in hysterical subjects is most essential.

CONCOMITANT STRABISMUS.

Concomitant strabismus is the name given to a form of squint which is caused by *excessive or defective convergence of the visual axes without any impairment of the conjugate movements of the eyes*.

It differs from paralytic squint in several ways (see p. 563).

1. The mobility of the affected eye is not markedly diminished in any particular direction. In many cases of convergent concomitant strabismus, however, there is defective outward movement of one or both eyes, the fields of fixation

being correspondingly limited. Similarly, in divergent strabismus there may be limited inward movement. When a muscular advancement is being performed, note is constantly made of the feebleness of the muscle. In some cases this may be a congenital malformation; in others, the result of disuse.

2. The primary deviation is equal to the secondary.

3. Diplopia is generally absent, but, when present, does not disappear in any particular part of the field of fixation, but the two images preserve a constant relation to one another in all positions of the eyes. Where diplopia is not complained of, it may frequently be elicited by placing before the non-squinting eye a red glass. This diminishes the distinctness of the true image, and lessens the contrast between it and the false image. Absence of diplopia may be explained in different ways, and probably results from different causes. In many cases the individual learns quickly the power of suppressing the image formed in the squinting eye; the younger the patient and the more amblyopic the eye, the more quickly is this power exercised. In other cases, though rarely, binocular single vision seems to be present together with a strabismus. A false macula is developed in the squinting, physiological rather than anatomical, with the result that the true macula of the fixing eye and this false macula of the squinting eye, which lies to the inner side of the anatomical macula, are 'corresponding' points. In such cases, if the eyes be put straight by operation, diplopia results, since the two anatomical maculae are not 'corresponding' points. This diplopia is, however, rarely permanent. A few cases of monocular diplopia have been recorded, where one eye has been previously lost, and where there is a history of a previous squint of the remaining eye. These cases may be explained by supposing that the secondarily acquired physiological macula retains its function.

4. There is no particular inclination of the patient's head.

Etiology and Pathology.—Concomitant squint may be convergent or divergent.

Convergent strabismus (internal strabismus) is as a rule associated with hypermetropia, although it occasionally occurs in emmetropic and myopic eyes. It generally commences in early life, usually during the fourth or fifth year. A patient

who is hypermetropic has always to use an excessive amount of accommodation in order to see objects clearly (see Refraction), and we have seen on p. 483 that the act of accommodation is naturally associated with that of convergence; hence the hypermetrope finds it easier to use the requisite amount of accommodation if he uses his convergence at the same time. The consequence of this is that the visual lines cross between the patient and the object looked at. If each eye deviated inwards to the same extent, it is evident that the image of an object placed in the middle line would fall in each eye to the inner side of the yellow spot, homonymous diplopia would be the result, and neither eye would see the object in its true position. We have seen, however, that the act of convergence is independent of the other conjugate movements. So that all the patient has to do, in order to see the object clearly, is to move both eyes to the right or to the left, so that (the same amount of convergence being maintained) the visual axis of one eye is directed to the object, while the other deviates inwards. Thus, supposing that the strabismus was such that, if it affected the two eyes equally, each eye would deviate inwards five degrees from its normal position, then, the one eye being directed to the object looked at, the visual axis of the other would deviate ten degrees from its normal position. The squinting eye in this case receives the image to the inner side of the yellow spot, and therefore projects it outwards; but as it is formed on a peripheral part of the retina, it produces a less intense visual impression than the image on the yellow spot of the other eye; hence the patient easily learns to disregard it, or, as it is termed, to 'suppress' it.

In the early stages the patient will often fix with either eye indifferently, and the squint is then said to be *alternating*. After a time, however, he acquires the habit of always fixing with the same eye, and the squint becomes *fixed* in the other. Even when the squint, however, has been fixed for many years, if the fixing eye be covered, the other can be made to fix the object, and the eye which is usually the fixing eye will squint; but, directly the eye is uncovered, it returns to its former position.

As long as the squint is alternating, each eye is used to the

same extent, but directly it has become fixed, the squinting eye ceases to be used, and its acuity of vision rapidly declines; this is unaccompanied by any ophthalmoscopic change, but when it has existed for any length of time it can only be improved by constant use of the eye, and even with this the visual acuteness can seldom be entirely restored; hence the importance of treating a squint directly it becomes fixed.

In the great majority of cases of convergent concomitant strabismus there is to be found a certain amount of *amblyopia*; that is to say, after any error of refraction has been corrected, the visual acuity of the squinting eye, and occasionally of both eyes, will be found to be subnormal, to a more or less considerable extent, even though the fundus oculi be normal in appearance. There has been much discussion as to which is primary, the amblyopia or the squint; but the fact that the amblyopia is occasionally present in both eyes points strongly to it being the cause of the strabismus, and not the reverse. As a general rule, the earlier the onset of strabismus the greater the amount of amblyopia. All infants are congenitally amblyopic, the full visual powers being only slowly acquired. Consequently any circumstance which interferes with the general development of the child may produce a detrimental effect on the visual apparatus. On questioning parents of squinting children some exciting cause is usually obtainable. Convulsions, teething, whooping cough, a fright, a fall, are among the exciting causes of a nerve-storm. The excess of convergence necessarily exercised by a hypermetrope to obtain binocular vision cannot be kept up by the disordered nervous system; one eye alone fixes the object, the more amblyopic of the two deviates. It is difficult to prove or to disprove that amblyopia is ever the result of a squint. It seems probable, however, that a congenital amblyopia may be increased in a squinting eye by disuse, though an eye which has once had good vision will probably retain it, even though a squint is present. I am firmly of opinion that much of the amblyopia of a squinting eye is frequently lost after the strabismus has been cured, especially in children under six years of age.

The field of fixation.—If the field of fixation of a patient

with convergent concomitant strabismus be taken (see p. 320), it will frequently be found to be defective in the outward direction, pointing to a weakness of the external rectus muscle. Not only is this the case in the deviating eye, but also sometimes in the fixing eye. Some have considered that this defect in the action of the external recti is a primary factor in the causation of convergent strabismus. In all probability, however, it is usually a secondary result of disease of the muscle.

Divergent strabismus (external strabismus), as a rule, occurs in association with *myopia*, although it is found in emmetropic and occasionally in hypermetropic eyes.

The connection between myopia and divergence is analogous to that between hypermetropia and convergence; in myopia the accommodation is used little, and in high degrees not at all; hence the patient finds it very difficult to use the great amount of convergence which would be required to obtain binocular vision at the close range at which he has to hold all objects. The difficulty of convergence is also increased by the elongated form of the globe, by which it is much less adapted than the more globular emmetropic eye for rotating in Tenon's capsule.

At first the divergence is only *relative*—that is to say, there is no actual divergence of the visual axes, but they are divergent *relative* to the point looked at; in other words, there is inability to converge. Later on the divergence becomes *actual*.

When an eye has ceased to be of use for visual purposes, whether from amblyopia, opacity of the cornea, or other causes, it frequently undergoes deviation, which as a rule takes place outwards.

Diagnosis and Measurement.—*To detect which is the squinting eye* is usually quite easy. We direct the patient to fix a small object, such as the tip of the index finger, held about half a metre in front of the eyes. One eye is then observed to be directed towards the object, and the other to be more or less deviating: this is called primary deviation. If the eye which the patient thus prefers to use be then covered by a disc of ground glass, the deviating eye will be observed to move before it can fix the object, and the covered eye will

now be seen, through the ground glass, to have deviated in a similar way to the first eye: this is called secondary deviation.

To find the amount of deviation.—1. This may be approximately effected by measuring the distance between two vertical lines, one passing through the middle line of the palpebral aperture, and the other through the centre of the pupil.



FIG. 172.—Measurement of the Angle of Strabismus.

Various instruments are made by which the distance between these two vertical lines can be measured in lines or millimetres.

2. The *angular measurement of strabismus* is more accurate than the above.

The angle of the strabismus is the angle which the visual axis of the deviating eye makes with the direction which it should have in a normal condition (Landolt). The

measurement of this may be effected by using the arc of the perimeter. The graduated arc $A O A$ (fig. 172) is placed horizontally. The deviating eye L is placed at the centre of the arc, and the patient is told to fix a distant object situated at O on the central radius. This he does with the normal eye R . Now the point O is that to which the deviating eye L would be directed in a normal condition. For all practical purposes it is sufficient to find the point x , on the *optic axis*, and to consider the angle $O L x$ as the angle of the strabismus.

To find this we pass the flame of a candle along the arc of the perimeter, keeping our own eye close to the candle, until the image of the latter is seen reflected from the centre of the cornea. The point x on the arc, at which this image is seen, is then read off, and we know the angle $O L x$.

The patient must not fix the white spot placed at the pole of the hemisphere of the perimeter. This would necessitate accommodation, and consequently would be liable to cause an increase in the amount of the deviation.

The Treatment of Concomitant Squint.—The goal at which all treatment should aim is, not the setting straight of the deviating eye, but the establishment of binocular vision. The younger the patient the more likelihood there is of succeeding; under six years of age operative treatment is not always necessary, and so should be postponed until other methods have been proved insufficient; in older patients an operation is almost always a necessity, and so should be performed earlier in the treatment, to be followed by the educative treatment.

In the first place, an account will be given of the treatment of a case of convergent concomitant strabismus soon after its onset—that is, the patient's age is probably under six years.

There are five definite steps in the treatment, all of which, however, may not be necessary in every case. These steps are: (1) the use of glasses; (2) the establishment of the power of fixation in the squinting eye; (3) the improvement of the vision of the squinting eye; (4) the establishment of the power of fusion; and (5) operation.

(1) *The use of glasses.*—In all cases the refraction of each eye should be carefully examined, and correcting glasses prescribed (see Refraction). It has been thought that children

under five are too young for spectacles ; but with a little persuasion, and with the help of a ribbon tied behind the head, glasses can be worn at the age of twelve months, or even earlier. It is necessary that the patient should in all cases be thoroughly atropinised, and the total error of refraction estimated. A correcting glass, less by $+0.5$ D spherical, should be ordered for constant use.

(2) *The establishment of the power of fixation.*—For this purpose the non-squinting eye must be effectually covered, the glasses being worn at the same time. The best method is to place on to the glass a large pad of some black material, or a gamgee pad may be fastened in front of the eye by means of strapping. This fixation power may often be obtained in the course of one or two months, if the pad is continually worn.

(3) *The improvement of the vision of the squinting eye.*—As soon as the power of fixation has been acquired, atropine must be ordered to be placed daily in the non-squinting eye only. This necessitates the child using the squinting eye for near vision, and in time (usually within three months) this eye becomes the working eye both for distant and near vision, the sight improving, and the previously straight eye now squinting. As soon as this result is obtained, step No. 4 must be commenced.

(4) *The establishment of the power of fusion.*—The best method that has been devised with this object in view is the systematic use of some form of stereoscope ; if the patient be a young adult, other methods (*vide infra*) may be combined with this. Javal was one of the first to insist upon the use of this instrument in the treatment of squint ; and Priestley Smith devised his ‘fusion-tubes’ and the ‘heteroscope,’¹ to enable the angle between the two halves of the instrument to be varied. Worth has recently described his ‘amblyoscope’ (fig. 173) as follows : ‘The instrument consists of two halves joined by a hinge at A. Each half consists of a very short brass tube joined to a longer tube at an angle of 120° . The diameter of the tube is $1\frac{1}{2}$ inches. Each half of the instrument is closed at A X by a flat oval plate of brass. Inside each half of the instrument, at A X, is an oval mirror.

¹ *Trans. Ophth. Soc.* vol. xviii. pp. 23 *et seq.*

G H, G H are the object-slide carriers. The object-slides consist of devices drawn on translucent paper and gummed on glass slips. At A B is a convex lens having a focal length of 5 inches—the distance of the reflected image of G H. A B, A B are slots into which other lenses from the trial case may be inserted if required. D E F is a brass arc with two slots in it—a short slot in which is the binding screw D, a long slot in which is the binding screw F. When the screw F is loosened, the two halves of the instrument can be brought together to suit a convergence of the visual axes up to 60° , or separated to suit a divergence of as much as 30° . When the screw F is

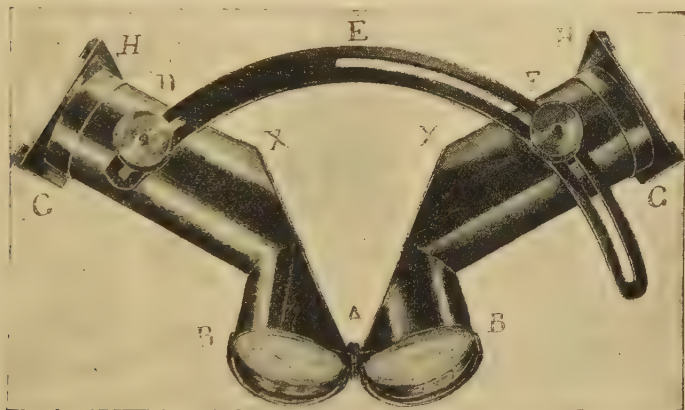


FIG. 173.—Worth's Amblyoscope.

tightened and the screw D in the short slot is loosened, an amplitude of movement of about 10° only is permitted.' Objects are used which will excite the interest of the child, and are arranged in three series: (a) those requiring merely simultaneous perception, *e.g.* a bird and a cage, a man and a horse; (b) those requiring fusion of images, *e.g.* two men, one with a right leg, the other with a left; and (c) those requiring the sense of perspective. The illumination of the objects can be varied by means of electric lamps adjustable on an upright stand (fig. 174).

The child should be seen twice a week for several weeks,

and soon takes great interest in the pictures. By gradually increasing the amplitude of fusion, and by altering the illumination of the two objects, a very powerful desire of fusion can be excited with patience and care.

(5) *Operation*.—Having established a good fusion power, and the deviation being stationary, an operation—the last step in the treatment—should be performed (see p. 585). It may

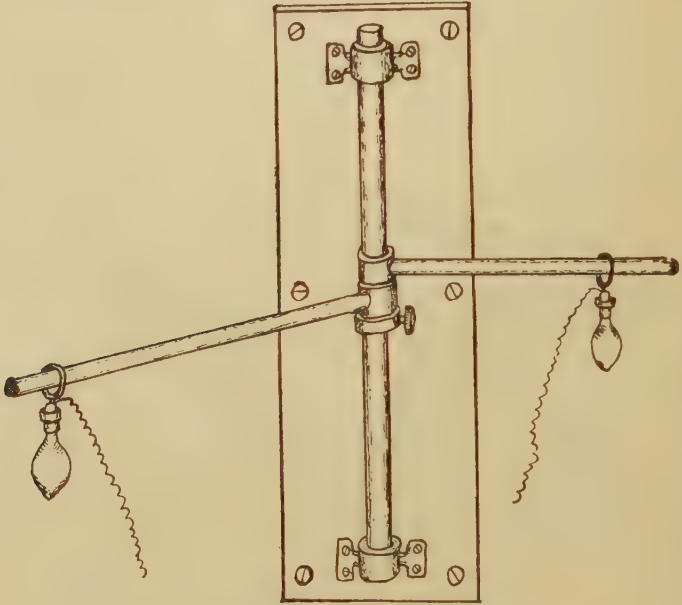


FIG. 174.—Illuminator for Worth's Amblyoscope.

be, however, that the previous steps have completely cured the squint.

A case of squint, however, may not present itself for treatment until the patient is more than six years of age, *i.e.* until several years have elapsed since its onset. The treatment here is somewhat modified. After an exact retinoscopy has been performed, and glasses ordered, it is well to operate without further delay, leaving the educational treatment till later. Having obtained complete or almost complete rectification of the deviation, three methods are available, all of which

should be patiently persevered with. These are: (a) the monocular shade, (b) bar reading, and (c) the systematic use of some form of stereoscope, such as the heteroscope or amblyoscope. In the second of these the bar is held between the eye and the paper, in such a way that part of each line is hidden from each eye, but if both eyes are used the whole can be read.

Divergent strabismus is very rarely, if ever, cured without the help of an operation.

Operative Treatment of Concomitant Strabismus.—There are two principal methods in operating for the cure of a concomitant strabismus. (a) By weakening the action of a muscle; (b) by increasing its power. The former is done by simply dividing its tendon—*tenotomy*; the latter, by advancing its tendon—*muscular advancement*.

(a) *Tenotomy.*—If the case is one of convergent or internal squint, tenotomy of the *internal rectus* muscle is necessary; if, on the other hand, it is a divergent or external squint, the *external rectus* should be divided.

The operation may be performed in one of two ways—either by exposing the tendon and then dividing it, or by the subconjunctival method of G. Critchett. In either case the tendon should be divided close to its attachment to the sclera.

Operation.—G. Critchett's method.—The instruments required are: (1) Speculum (fig. 82); (2) Fixation forceps



FIG. 175.—Strabismus Scissors.

(fig. 43) ; (3) Squint hook (fig. 41) ; (4) Blunt-pointed straight scissors (fig. 175).

The patient lies supine; the surgeon stands on the patient's right, and his assistant opposite to him. The speculum is introduced, and the lids widely separated; the surgeon pinches up a fold of conjunctiva and subconjunctival tissue with the fixation forceps on the same side as the muscle he is about to divide: if on the inner side, midway between the cornea and caruncle; if on the outer, between the cornea and outer canthus; always, however, on the level with the



FIG. 176.—Convergent Strabismus.



FIG. 177.—After Operation.

lower margin of the tendon. With the scissors in the right hand held almost perpendicularly, he divides these structures, and the capsule of Tenon. The latter is known to be opened when the point of the scissors suddenly sinks deeper into the wound; if there is any resistance offered, the capsule is probably intact. That membrane being opened, and the white shiny sclerotic exposed, the hook, with its point downwards, is introduced into the wound, and, being turned towards the globe, the concavity of the hook is made to sweep upwards over the convexity of the eyeball, and beneath the rectus tendon, at the upper

border of which the point will be seen projecting beneath the conjunctiva. On now drawing the hook forwards, the tendon will occupy its concavity about 5, 6, or 7 mm. from the lateral margin of the cornea, and the pull upon it will rotate the globe to the opposite side.

It is necessary to be careful to open the fibrous capsule of Tenon, otherwise the hook will not pass beneath the tendon, but between it and the conjunctiva ; if this has occurred it will pass right up to the corneal margin instead of being arrested by the muscle. The forceps is now relinquished, and the hook passed to the left hand, by which it is held parallel to the nose, while some traction is made in a forward direction so as to tighten the

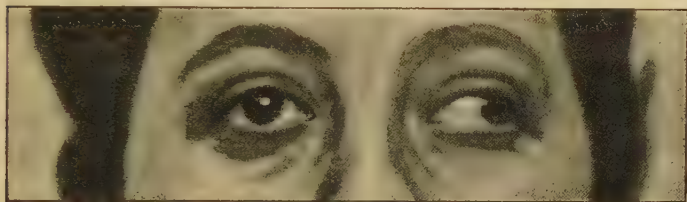


FIG. 178.—Divergent Strabismus.



FIG. 179.—After Operation.

tendon and render it accessible to the scissors. The latter are now to be passed into the wound *between the hook and the eye* ; in doing this the blades should be slightly open, so that one passes in front and the other behind the tendon, which must be divided *close to the sclerotic* by two or three snips. When this has occurred, the hook can be drawn forwards right up to the margin of the cornea. It should, however, be introduced a second time, to ascertain if any strands of tendon have escaped division.

Where a considerable effect is desired, the scissors should be passed between the conjunctiva and the globe in front of the rectus before the tendon is divided, and during the division of the latter the incision of the surrounding capsule of Tenon should be more extensive than in a simple tenotomy. It must be remembered that the insertion of the external rectus is farther back on the globe (6·9 mm.), and that it is in closer apposition to the latter, and consequently more difficult to hook than the internal rectus.

The main difficulties in the division of either of these tendons are in opening and introducing the hook into Tenon's capsule, and in cutting through the tendon without pushing it off the end of the hook with the scissors.

(b) *Muscular advancement or adjustment* signifies the detachment of a tendon from its insertion in the sclerotic, and bringing it forward in such a manner that it may become adherent at a point in front of its original position. By this means its power in the rotation of the globe is increased.

The operation which I have found most successful is Worth's modification of Prince's; it requires speculum

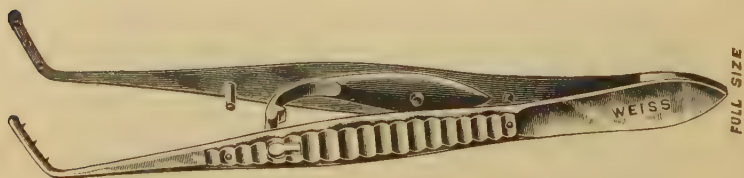


FIG. 180.—Prince's Catch-Forceps.

(fig. 82), blunt-pointed straight scissors (fig. 175), Prince's catch-forceps (fig. 180), two curved needles each threaded with a specially prepared black silk suture,¹ and a needle-holder. Worth stands at the head of the patient; personally, I prefer the right side whichever eye I am operating upon. A straight

¹ Worth uses thick black silk made for sewing boots, marked 'No. 24, W. H. Staynes & Smith, Belgrave Gate, Leicester.' This is boiled in water and then dried. It is drawn through a very hot mixture composed of three parts white beeswax and five parts white vaseline, then wound on a glass reel, and kept ready for use in a sterilised glass jar.

vertical incision half an inch long is made in the ocular conjunctiva with the scissors; the middle of the incision should be opposite the centre of the cornea, and close to its lateral margin. Tenon's capsule is similarly incised. The female blade of Prince's forceps is now passed under the tendon, and the conjunctiva and capsule are drawn forwards between the two blades of the forceps, which are now closed. The tendon is then divided at its insertion. In the next place, the sutures

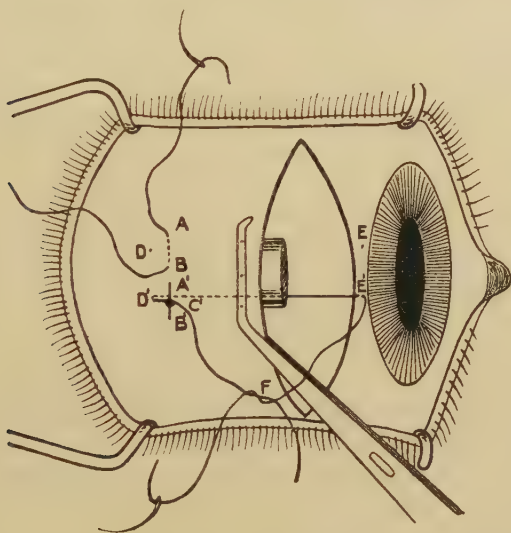


FIG. 181.—Worth's Operation for Muscular Advancement.

must be passed. The positions of entrance and exit are shown in fig. 181. One is passed from without inwards through conjunctiva, capsule, and muscle at B, and brought out again through the same structures at A. The other suture is similarly entered at B', and brought out at A'. The two ends of each suture are knotted together as at C'. The end armed with the needle is again entered, as at D', passed through conjunctiva, capsule, and muscle, and is carried horizontally beneath the forceps, to be passed through the episcleral tissue close to the corneal margin, as at E'. The

forceps is removed by cutting through the muscle, capsule, and conjunctiva immediately behind the blades. The two ends of each suture are now tied, so as to obtain the required rotation of the eyeball.

(c) *Capsular advancement*.—This is a modified form of the last operation performed by some surgeons, which I find useful in cases where but little is required to correct an external deviation or a slight convergence still existing after previous tenotomy of the rectus internus.

The operation is conducted in a similar manner and with the same instruments as in muscular advancement; the chief difference lies in *not dividing the tendon*. It is not necessary to use Prince's forceps, though the sutures are more easily passed when the structures are held together within its blades. The sutures are passed as before. When the sutures are tied the tendon is folded upon itself; this constitutes the shortening process, and is, therefore, a muscular advancement, and not, strictly speaking, an advancement of the capsule only, as the title implies.

The after-treatment.—Simple tenotomy is rarely attended by serious reaction. A sterilised pad of gamgee tissue and bandage should be worn for twelve hours and then discarded. The subconjunctival extravasation of blood usually disappears in the course of two weeks; but if severe, as sometimes happens when vomiting takes place immediately after the operation, its absorption may require a longer period than this.

Where ametropia exists, the correcting glasses should in all cases be worn from the time of the operation.

Muscular or capsular advancement is followed by pain and swelling, which may be considerable, and usually extend over a few days. An aseptic dry dressing should be worn, and the patient kept in bed during the reaction. It is well to bandage both eyes for from two to four days. When the eye that has not been operated upon is uncovered, it should be atropinised to prevent accommodation. The sutures should be removed on the seventh or eighth day. All dressings may be discarded on the day following this, and glasses should now be worn.

Complications and sequelæ.—The operation of tenotomy is easily performed. Cases have occurred where, during the

actual division of the tendon, the sclerotic has been cut through, vitreous presenting. Due care, however, reduces this risk to a minimum. Considerable subconjunctival hæmorrhage sometimes necessitates the making of a counter-opening through the conjunctiva above the tendon. Hæmorrhage may also occur into Tenon's capsule.

The following sequelæ of the operative treatment of strabismus may occur :

1. Suppuration, with or without orbital cellulitis. If strict antiseptic and aseptic precautions are taken, there is little risk of this.

2. Sinking of the caruncle, with enlargement of the palpebral aperture at the inner canthus. This is sometimes seen after a tenotomy of the internal rectus. The receding capsule draws backwards the caruncle, and produces slight deformity. Some operators put a stitch into the conjunctival wound to obviate this effect.

3. Exophthalmos. Slight protrusion of the eye is sometimes found after a tenotomy. It is rarely permanent.

4. Permanent thickening of conjunctiva, with the formation of a small nodule of granulation tissue, sometimes happens as the result of an advancement. Removal of any redundant conjunctiva at the time of the operation is usually successful in preventing this occurrence.

5. Diplopia. This, though very annoying to the patient, is only temporary, and is usually the prelude to the acquirement of binocular vision.

6. Weakness of movement of the eye in the direction of the tenotomised muscle. Thus, in some cases of tenotomy of one or both internal recti, the power of convergence is somewhat impaired.

7. Divergence. This is an occasional late result of internal tenotomy, if the tenotomy is performed on an eye which has a considerable degree of amblyopia. It may be remedied by a subsequent advancement.

8. Alteration in any pre-existing astigmatism. The bringing forward or setting back of a muscle will tend to alter the curvature of the cornea in the corresponding meridian.

Tenotomy versus advancement.—There is considerable difference of opinion as to the indications for tenotomy; in fact, some surgeons go so far as to say that this operation should seldom, if ever, be performed. Advancement is preferred on the ground that in the operation a weak muscle is strengthened, while in tenotomy a normal or slightly weakened muscle is further weakened; the opponents of tenotomy also urge as evidence in favour of their views that this operation produces more permanent disfigurement than does advancement. They also cite instances where a convergent strabismus has, by a free internal tenotomy, been converted into a divergent strabismus. My own opinion is that the vision of the squinting eye is the best guide in deciding the nature of the operation. If a considerable degree of amblyopia is present, a single or double advancement should be performed, rather than a free tenotomy. Where the amblyopia is moderate only, a combined tenotomy and advancement is what I usually advise. If the amblyopia is only slight, I generally first try the effect of a single simple tenotomy, to be followed, if necessary, by an advancement of the antagonist.

Capsular advancement of the rectus internus is very useful in those cases where a blind eye deviates slightly outwards from disease, or where slight divergence follows an operation for a convergent squint.

The choice of an anæsthetic.—The value of the local anæsthetic cocaine cannot in my mind be over-estimated in the performance of these operations. Its superiority over a general anæsthetic lies in the fact that the squint can be corrected at one operation; for the patient, being conscious, can use the eyes during the operation, and so help the operator by different directions of regard; and if any deviation still exists it is recognised and can be corrected by a further operation, either by a tenotomy in the other eye or advancement of the antagonist on the same eye. Under a general anæsthetic the eyes usually diverge, and a very marked convergent squint will often appear divergent under chloroform. Again, cocaine produces complete local anæsthesia: no pain is, as a rule, felt beyond a slight unpleasant pull on the tendon when the hook is introduced. In children

under twelve, or in highly nervous individuals, a general anæsthetic is usually required.

The *use of prisms* is recommended by some surgeons (Du Bois Reymond, Javal) as a means of cure for concomitant squint. The strength of the prism should be one or two degrees less than the angle of the strabismus, so that the patient can practise fusion of the double image by the use of the two eyes together. This method can be of service only in those very rare cases in which there is still binocular vision.

HETEROPHORIA.

Heterophoria, formerly wrongly termed *muscular insufficiency*, consists in the inability to maintain binocular vision beyond a short period of time. With a squint, the patient cannot command binocular vision at all; with heterophoria, he can do so for a short while. The causes are most frequently errors of refraction, though general weakness brought about by anæmia, nervous exhaustion, and the like, is often attended by this condition with symptoms allied to asthenopia, in which no error is to be found in the patient's refraction, the cause being a faulty muscular innervation; more rarely there is definite anatomical muscular feebleness.

The movements usually at fault are those of convergence and divergence; for though the recti interni govern the immediate act of convergence, their action is regulated by the relaxation of their antagonists, the externi; also the return to parallelism is brought about by the contraction of the latter similarly modified by the relaxation of the interni.

Deficient nutrition of the externi, or excessive nutrition of the interni, will allow a deviation inwards of one eye when parallelism can no longer be excited by binocular vision—*insufficiency of divergence*, or *excess of convergence*.

Similarly, deficient nutrition of the interni, or excessive nutrition of the externi, will, after the eyes have been fixing a near object for a short while, be attended with deviation of one eye outwards away from the point of fixation—*insufficiency of convergence*, or *excess of divergence*.

The heterophoria attending refractive errors is in many

instances due to a disturbance in the balance between accommodation and convergence (see p. 483), but in others there can be no doubt that a muscular weakness, whether congenital or acquired, exists, which gives rise to symptoms allied to asthenopia, and they are expressed by many as 'muscular asthenopia.' Myopia is usually attended with insufficiency of convergence; hypermetropia, on the other hand, with insufficiency of divergence.

In insufficiency due to other causes, weakness of convergence is more often witnessed, the eyes tending to turn outwards into the position of natural rest.

The examination of heterophoria.—(a) *Maddox's rod test.*—This is both qualitative and quantitative, and is the most useful method of estimating heterophoria. The rod (fig. 182) now in use is a composite one, made up of a parallel series of small red glass rods cemented together and mounted in a vulcanite disc. The tangent scales of Maddox (fig. 183) consist of a vertical and horizontal scale so placed as to cross each other at right angles with their zeroes coinciding, marked with figures representing degrees ranging from the central

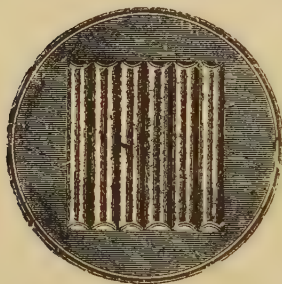


FIG. 182.—Maddox's Rod.

point, and printed red to the left and black to the right. A small lamp is placed at the centre of the scales.

The patient should be placed at a distance of 5 metres from the scales, with the rod placed first horizontally in front of one eye, and a green or blue glass in front of the other eye. If there is muscular equilibrium, the vertical red line will pass through the centre of the green or blue flame (*orthophoria*). If, however, the line lies to one side of the flame, there is a latent deviation (*heterophoria*), either latent divergence (insufficiency of convergence, or excess of divergence—*exophoria*) or latent convergence (insufficiency of divergence, or excess of convergence—*esophoria*); in the former the diplopia will be crossed, in the latter homonymous. Secondly, the rod is placed vertically; and if there is no heterophoria, the hori-

zontal red line will pass through the centre of the flame. If, however, the line lies above or below the flame, there is a latent deviation (*hyperphoria*): left hyperphoria if the image in the left eye is below that in the right, and right hyperphoria if the opposite position exists.

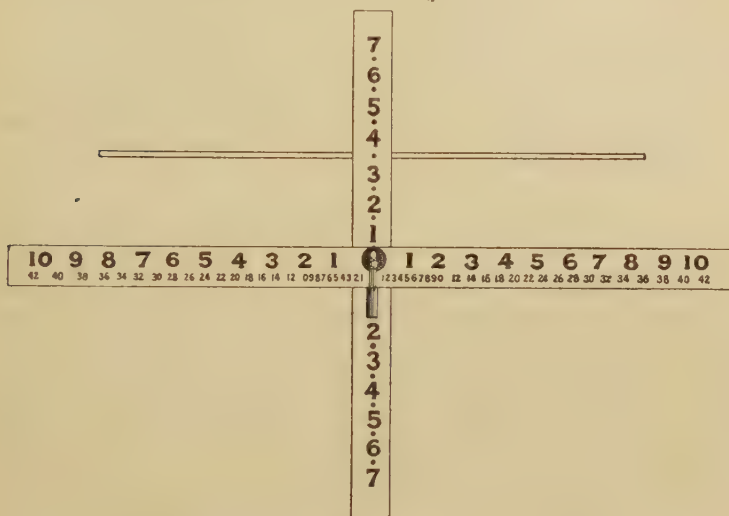


FIG. 183.—Tangent Scales of Maddox.

In both cases the distance between the images, as shown by the scales, is the measure of the degree of heterophoria.

(b) *Prism test*.—This test was first introduced by the elder Graefe, and was used to estimate heterophoria both for distant



FIG. 184.—Maddox's Test (reduced).

and near vision. Now, however, the prism is chiefly used for near vision only, and is employed by Maddox in the following way. The prism is a single square one with a deviation of 6° . A small tangent scale (fig. 184) replaces the large scale of the rod test. It is graduated in degrees and also metre-angles on either side of the central zero point, the markings being in

red to the left and in black to the right. A vertical arrow is placed at zero, and a printed sentence underneath the scale for which the patient is required to accommodate.

The patient is placed at a distance of 25 cm. from the card, with the prism before the right eye with its edge upwards. He is required to say to which figure, and whether black or red, the lower of the two arrows he sees, points. If the lower arrow is to the right, the diplopia is crossed, and exophoria is present; if to the left, the diplopia is homonymous, and esophoria is present. It must be remembered that at 25 cm. a physiological exophoria is present varying from 0° to 6° .

(c) Another prism test by the same author is in use, and consists of two prisms attached base to base. This, properly adjusted in front of one eye, will produce monocular vertical diplopia. A point of light looked at through this double prism is seen as two, and if the other eye is in use it will see the true light midway between these false images. If the true image appears to be upon an imaginary line drawn between the two false ones, there is orthophoria; if it does not, there is heterophoria or latent deviation.

Treatment.—The first step to be taken in the treatment of heterophoria is the removal of the cause; this in most instances is all that is required. Correct any existing ametropia, and support the general health, by which means the general nervous system is improved. Should it happen that no refractive error is present, and that there is no constitutional depression, the conclusion arrived at is that there exists an inherent weakness of innervation of the abducting or adducting muscles of the eyes. In such cases prisms (p. 483) may relieve the symptoms: if there is esophoria, prisms with their bases outwards, if exophoria prisms with their bases inwards, will allay discomfort. In hyperphoria, a prism with its base down should be placed before the hyperphoric eye, and one with its base up before the other eye. ~~If there~~ should be present any heterophoria after the proper correction with accurately centred lenses and after improving the general muscular tone, the correcting prism should be constantly worn—alone if no error of refraction is present; or, if there is an

error, in combination with the proper sphere by the decentration of the latter. When heterophoria causes discomfort, Maddox has suggested the following rules as guides in the prescribing of prisms :

‘Three-quarters of a persistent *hyperphoria* should be corrected by prisms, or by decentering.

‘Two-thirds of a distant, or the whole of a near *esophoria*.

‘Half or a third of a distant, and a quarter of a near *exophoria*.’

In insufficiency due to general muscular weakness, besides constitutional treatment local help may be afforded by the daily use of prisms so as to practise abduction and adduction. Some object is fixed (6 metres distant) and prisms of increasing strengths are used, an endeavour being made to prevent diplopia by maintaining binocular vision (Dyer’s treatment) ; to prescribe prisms for the mere relief of distress in such cases is not justifiable. In the worst cases of insufficiency, in which binocular vision cannot be maintained for a longer period than two or three minutes, it may be necessary to weaken the opposing muscle by tenotomy or to strengthen the defective muscle by the advancement of the capsule near its insertion, or even by advancement of the muscle itself.

NYSTAGMUS.

Nystagmus is an oscillating movement of the globes, produced by the involuntary and jerky contractions of the ocular muscles. It is commonly associated with some serious defect of vision which has existed from very early life, without absolute loss of sight, such as opacities of the cornea after purulent ophthalmia, pyramidal and all forms of congenital cataract, albinism, choroido-retinitis, and other affections. The cause of nystagmus developed in infancy probably depends upon an imperfect development of the co-ordinating centres governing the conjugate movements of the eyeball. The natural power of fixation is an acquired one, and results from the perception of perfect retinal impressions. Defective sight from early childhood is an impediment to this physiological acquirement. The little light that is perceived causes an imperfect attempt to be made to use the eyes ; an irregular

action of the co-ordinating centre is the result. Absolute blindness from birth is rarely, if ever, attended with nystagmus.

Nystagmus is often developed in adult life amongst persons who work in coal-pits (*miners' nystagmus*). It occurs in about 5 per cent. of all miners. That this variety is due to a faulty position of the body, head, and eyes, is strongly advocated by Snell.¹ Other observers attribute the disease to paucity of light. I am inclined to look upon the former as the exciting and the latter as the predisposing cause of this affection. The eyes turned obliquely upwards soon tire from the insufficient controlling action of the co-ordinating centre, weakened from want of external stimuli; for the functional activity of that centre is to a great extent maintained by external influences. Other predisposing causes of this affection have been considered to be errors of refraction, especially hypermetropia and hypermetropic astigmatism, and any impairment of the general health. Workers in occupations other than mining, where there is unusual strain on the elevators of the eyeball, occasionally acquire nystagmus. These are compositors, platelayers, metal rollers, employes in glass factories, plank-cutters, &c.

In certain diseases of the central nervous system nystagmus is a frequent symptom; as in insular sclerosis, hereditary ataxia, meningitis, hydrocephalus, intracranial new-growths especially cerebellar tumours (Gowers), cerebellar abscess, and primary spastic paraplegia. It is occasionally found in syringomyelia, ataxia paraplegia, and progressive muscular atrophy. It is a curious fact that it is never seen in paralysis agitans.

The oscillatory movement is almost invariably bilateral, and may take place in any direction; it may be horizontal, vertical, oblique, or rotatory. The horizontal movement is that most frequently met with. The movements vary greatly in rapidity and extent in different cases, and even in the same case. In the miner, for instance, it often only takes place when he is in the stooping posture; in this form of nystagmus the number of oscillations per minute varies from 60 to over 100.

Some few cases of voluntary nystagmus have been re-

¹ *Trans. Ophth. Soc.* vol. iv. p. 315; vol. xvi. p. 305.

corded. Grimsdale¹ has recorded four cases of nystagmus which occurred only on covering one eye, or on producing diplopia by means of prisms. On vision becoming binocular, the nystagmus disappeared.

Treatment fails to cure the affection. The vision should when possible be improved by glasses.

THE INTRA-OCULAR MUSCLES.

The internal muscles of the eye are the ciliary muscle, the sphincter pupillæ, and the dilator pupillæ. The anatomy and physiology of these muscles have been described in Chapter VI.

Pathological variations in the size of the pupil fall under two chief headings—(i) *Vascular changes*; and (ii) *Disturbed innervation*.

(i) If the iridic vessels are distended with blood, the pupil will be contracted, as in congestion and inflammation of the iris; also in plethoric individuals from general vascular engorgement. If the iridic vessels are contracted and the iris anæmic, the pupil will be dilated, as in chlorotic and anæmic subjects. Mydriatic drugs act partly by constricting the vessels. The pupillary dilatation in diseases attended with increased intra-ocular tension may also in part be due to this vascular change.

(ii) Mydriasis from disturbance in the nervous mechanism may be caused by an irritative lesion in the cervical sympathetic cord or in the spinal cord involving the cilio-spinal centre. Mydriasis is also present in epidemic cerebro-spinal meningitis, internal spinal meningitis, and occasionally in tabes dorsalis. A destructive lesion of the third nerve nucleus, the third nerve trunk, or the lenticular ganglion, will cause dilatation of the pupil on the same side as the lesion. This is seen in the later stages of tubercular meningitis; it is also present in cases of cerebral hæmorrhage. Any loss of continuity or conductivity in the afferent portion of the reflex arc will be attended with pupillary dilatation; a destructive lesion involving both nerves will produce bilateral mydriasis; a lesion of one

¹ *Trans. Ophth. Soc.* vol. xvi. p. 328.

optic tract will be attended with the *hemiotic pupil* (p. 600) ; destruction of one optic nerve or any marked impairment of retinal activity, as optic atrophy, tumour of optic nerve, retinal atrophy, retinal detachment, and the like, will give rise to dilatation of the pupil, with loss of direct light-reflex, without interfering with the consensual light-reflex.

Amblyopia from organic disease of the optic nerve, with perhaps no ophthalmoscopic signs, may therefore be distinguished from true functional amblyopia, and also from malingering, in which conditions the pupil phenomena are normal. In slight cases of organic disease, however, there may be merely an alteration in, not a loss of, pupil activity. If the stimulus is prolonged, the contraction is not sustained, the pupil of the affected eye either oscillating or becoming dilated.

Mydriatic drugs (p. 191) act chiefly by paralysing the terminal ends of the third nerve, also, in part, by their direct action on the walls of blood-vessels, and by direct stimulus to the fibres of the dilator pupillæ. Cocaine acts mainly by stimulating the sympathetic.

Direct paralysis of the sphincter pupillæ may be caused by a blow upon the eyeball (p. 633).

Miosis may be due to a destructive lesion of the cervical sympathetic cord or the cilio-spinal centre. Thus it is frequently found in tabes dorsalis and in syringo-myelia. It may also result from an irritative lesion of the nucleus or trunk of the third nerve, as in the first stage of meningitis, in pontine lesions, and in other progressive central degenerations. The miotic drugs (p. 179) act by stimulating the peripheral ends of the oculo-motor nerve.

A *hemiotic pupil* is one that, in certain cases of hemianopsia, does not contract to a strong light thrown upon the blind half of the retina, but reacts readily to a light impinged upon the seeing half of that membrane. It is a strong evidence in favour of a destructive lesion involving the afferent portion of the reflex arc, and so indicative of one at or in front of the optic centre. If a light thrown on the functionless half of the retina calls forth a ready pupillary response, the lesion must be beyond the optic centre, *i.e.* in the internal capsule,

optic radiation, or cortex cerebri. This symptom is obviously of diagnostic value only in homonymous hemianopsia (p. 331). A practical demonstration of this phenomenon is by no means easy.

The **Argyll Robertson pupil** is the phenomenon often witnessed in certain progressive degenerative lesions of the spinal cord and brain, as tabes dorsalis, general paralysis of the insane, and, in rare instances, insular sclerosis. The pupil reacts to accommodation, but not to light—*reflex iridoplegia*; both the direct and consensual reflexes are lost, unless, as is occasionally the case, the phenomenon is unilateral; in such a case the pupil of the unaffected eye will respond to the indirect stimulus (Jessop).¹ The probable explanation of the phenomenon is that degeneration has taken place in the path between the third nerve nucleus and the optic centres.

Cycloplegia, or paralysis of the ciliary muscle, is found in all degrees of severity. Its chief causes are syphilis, diphtheria, influenza, injury, the use of certain therapeutic agents such as atropine, and poisoning by certain alkaloids such as belladonna. A destructive lesion of the anterior part of the third nerve nucleus produces cycloplegia. It is usually associated with a similar affection of the sphincter pupillæ, although it is occasionally found alone, as after certain cases of diphtheria. It is usually seen in paralysis of the third nerve. There is loss of the power of accommodation (p. 476), the pupil is generally dilated. Functional troubles, similar to those of presbyopia, are experienced, and if the eye is hypermetropic there is deficiency in both near and distant vision. On the other hand, in myopia the troubles in near vision are much less. The prognosis is in many cases favourable, especially when diphtheria or syphilis is the cause. Tonics or antisyphilitic remedies constitute the treatment.

Loss of accommodation from senile changes (presbyopia), and paralysis from the use of therapeutic agents such as atropine, homatropine, duboisin, hyoscyamine, &c., will be found discussed in the chapter on Refraction.

¹ *Trans. Ophth. Soc.* vol. xi. p. 179.

CHAPTER XVI.

THE ORBIT.

ANATOMY—CELLULITIS—PERIOSTITIS—EDEMA—THROMBOSIS OF CAVERNOUS SINUS—INTRA-ORBITAL HÆMORRHAGE—EMPHYSEMA—DISTENSION OF FRONTAL SINUS—MENINGOCELE—EXOPHTHALMIC GOITRE—TUMOURS—NEUROMA—LIPOMA—FIBROMA—CYSTS—HYDATID CYSTS—CYSTICERCUS CELLULOSÆ—EXOSTOSES—SARCOMA—CARCINOMA—PULSATING EXOPHTHALMOS—TEMPORARY EXOPHTHALMOS—ERECTILE OR CAVERNOUS TUMOUR.

Anatomy of the Orbit and its Contents.—The orbit is a pyramidal-shaped cavity with a quadrilateral base, and has a depth of 5 cm. Its base is directed forwards and outwards, and is outlined by the frontal, malar, and superior maxillary bones. Its *roof* is mainly horizontal, and is formed by the orbital plate of the frontal bone, and, posteriorly, by the lesser wing of the sphenoid. At the antero-external corner is the fossa for the lachrymal gland. The anterior margin of the roof is interrupted at the junction of its inner and middle thirds by the supra-orbital notch or foramen, through which pass the supra-orbital vessels and nerve. The *outer wall* of the orbit forms almost a right angle with its fellow of the opposite side; it is made up of the great wing of the sphenoid, and, anteriorly, the malar bone. The temporal and malar branches of the temporo-malar nerve pierce the malar bone, the former passing to the temporal fossa, the latter appearing on the cheek. The outer wall is separated from the roof by the *sphenoidal fissure*, whose direction is outwards and slightly upwards. This fissure, which is widest at its inner extremity, leads into the middle cranial fossa, and transmits the third nerve, the fourth nerve, the three divisions of the fifth nerve, the sixth nerve, the two ophthalmic veins, the recurrent branch of the ophthalmic artery, the orbital branch of the middle meningeal artery, and sympathetic filaments from the cavernous plexus. Between the outer wall and the floor, for the posterior two-thirds, is the *spheno-maxillary fissure* leading into the spheno-maxillary fossa, and transmitting the superior maxillary division of

the fifth nerve and its temporo-malar branch, the infra-orbital vessels, twigs from Meckel's ganglion, communicating veins from the inferior ophthalmic vein to the pterygoid venous plexus, and lymphatic vessels to the deep facial glands. These two fissures are closed by membrane, that of the spheno-maxillary fissure being largely composed of unstriped muscular fibres, frequently termed 'Müller's muscle.'

The *floor* of the orbit slopes downwards and outwards. It is chiefly formed by the orbital plate of the superior maxilla, the orbital plate of the palate bone appearing only at the extreme posterior part; at the outer angle is the malar bone. The floor contains the infra-orbital groove for the nerve and vessels of the same name; this groove is bridged across by a membrane composed chiefly of involuntary muscular fibres. The *inner wall* is vertical, and parallel to its fellow. From before backwards the following bones enter into its composition: the frontal process of the superior maxilla, the lachrymal, the os planum of the ethmoid, and the body of the sphenoid. Between the inner wall and the roof are placed the anterior and posterior ethmoidal foramina for the ethmoidal vessels; through the former the nasal nerve also passes. At the *apex* of the orbit is placed the optic foramen, which is situated above and internal to the inner end of the sphenoidal fissure. Through this foramen pass the optic nerve and the ophthalmic artery.

The most important relations of the orbital walls are with regard to the air-sinuses, which surround it on all sides except externally. The roof separates the orbital contents from the frontal sinus, and, internally, from the ethmoidal cells. The floor forms the roof of the antrum of Highmore, and is also in close relationship internally with the lower ethmoidal cells. The inner wall contains the fossa for the lachrymal sac which separates the orbital cavity from the middle meatus of the nose, and, more posteriorly, this wall is the outer wall of the anterior ethmoidal cells. The orbit is, near the apex, closely related to the sphenoidal air-cells.

The interior of the orbit is lined by periosteum. This is continuous with the dura mater through the sphenoidal fissure, and with the periosteum on the exterior of the skull round the margins of the base of the orbit, and through the spheno-maxillary fissure. The palpebral ligaments are closely connected with it.

The orbit contains the following structures: the eyeball and its optic nerve, the extra-ocular muscles and the capsule of Tenon, the ophthalmic artery and its branches, the orbital branch of the middle meningeal artery, the two ophthalmic veins and their tributaries, lymphatic vessels, the third, fourth, ophthalmic and superior

maxillary divisions of the fifth, and sixth cranial nerves, sympathetic nerves from the cavernous plexus, the lachrymal gland and its ducts, and a considerable quantity of fat. The anatomy of many of these structures has been considered in former chapters, but a brief account must here be given of the vessels and nerves, as well as of the orbital fat.

Ophthalmic Artery.—This vessel enters the orbit through the optic foramen to the outer side of the optic nerve. It almost immediately crosses the nerve, accompanied by the nasal nerve and the superior ophthalmic vein. Running forwards along the inner side of the optic nerve, it divides at the anterior and inner part of the orbit into its terminal branches, the frontal and nasal arteries. Its branches are as follows :

- | | |
|------------------------------|--------------------------------------|
| 1. Recurrent. | 6. Anterior and Posterior Ethmoidal. |
| 2. Posterior Ciliaries. | 7. Supra-orbital. |
| 3. Muscular. | 8. Superior and Inferior Palpebral. |
| 4. Arteria Centralis Retinæ. | 9. Nasal. |
| 5. Lachrymal. | 10. Frontal. |

1. *Recurrent branch.*—This passes backwards through the sphenoidal fissure to reach the middle cranial fossa.

2. *Posterior ciliary arteries.*—These are described on p. 175.

3. The *muscular branches* supply the extra-ocular muscles, and give off the anterior ciliary arteries (see p. 175).

4. The *arteria centralis retinæ* enters the optic nerve 12 mm. behind the eyeball, and running forwards, reaches the papilla. For its further course see p. 248.

5. The *lachrymal artery* runs along the outer part of the orbit, and gives off branches to the lachrymal gland. It supplies a palpebral branch to each eyelid, anastomosing with the palpebral branches from the ophthalmic artery itself. It also sends a branch which anastomoses, in the neighbourhood of the apex, with the orbital branch of the middle meningeal artery.

6. The *anterior and posterior ethmoidal branches* soon leave the orbit through the ethmoidal foramina.

7. The *supra-orbital artery* arises from the main trunk as it is crossing the optic nerve.

8. The *palpebral branches* are given off to supply the inner part of each eyelid.

9. The *nasal terminal branch* leaves the orbit above the internal tarsal ligament, by piercing the palpebral fascia.

10. The *frontal terminal branch* pierces the palpebral fascia at the upper and inner part of the orbit.

The **orbital branch of the middle meningeal artery** is the only artery found in the orbital cavity which is not a branch of the ophthalmic artery. It is a small twig which enters by the sphenoidal fissure, and anastomoses with a branch of the lachrymal artery.

The **orbital veins** are two in number, the superior and inferior ophthalmic veins.

The *superior ophthalmic vein* commences at the root of the nose, where it communicates with the angular vein, and so with the facial vein. It passes backwards along the inner wall of the orbit, crosses the optic nerve near the apex, and, running between the two heads of the external rectus muscle, leaves the orbit through the sphenoidal fissure to enter the anterior part of the cavernous sinus.

The *inferior ophthalmic vein* communicates with the pterygoid venous plexus through the sphenomaxillary fissure. It lies deep to the optic nerve, and leaves the orbit through the sphenoidal fissure, having passed between the heads of the external rectus. It too ends in the cavernous sinus.

The smaller veins of the orbit correspond to the arteries, some entering the superior, others the inferior ophthalmic vein; the arrangement is variable. There is a constant communication between the two large veins; they may join or end separately in the cavernous sinus. The central vein of the retina opens directly into the sinus, but usually it is connected with one of the main vessels. The orbital veins are destitute of valves.

Orbital Lymphatic Vessels.—The orbit is not furnished with many lymphatic vessels, since most of the lymph circulation takes place through Tenon's capsule and the lymph spaces of the optic nerve. What vessels exist are efferent. Some pass through the sphenomaxillary fissure to the deep facial glands; some end in the zygomatic glands on the posterior part of the buccinator muscle; whilst others pass to the internal maxillary glands situated on the anterior part of the wall of the pharynx.

The Orbital Nerves.—The nerves of the orbit are very numerous. They comprise five cranial nerves—namely, the second or optic, the third or oculo-motor, the fourth or trochlear (patheticus), the ophthalmic and superior maxillary divisions of the fifth or trigeminus, and the sixth or abducent—and sympathetic filaments from the cavernous plexus.

The *optic nerve* has been described on p. 240.

The *oculo-motor nerve*, as it enters the orbit through the sphenoidal fissure, divides into superior and inferior divisions. Both

pass between the two heads of the rectus externus. The superior division sends branches to the superior rectus and the levator palpebræ superioris, both entering the muscles on their ocular surface. The inferior division supplies the inferior and internal recti on their ocular surface, and the inferior oblique at its posterior border; the filament to the last-named muscle gives to the ciliary muscle its short root.

The *trochlear nerve* passes into the orbit through the sphenoidal fissure above the muscles, and enters the superior oblique on its orbital surface.

The *ophthalmic division of the trigeminal nerve* divides into the lachrymal, frontal, and nasal nerves before reaching the orbit. The *lachrymal nerve* enters through the outer angle of the sphenoidal fissure, passes forwards along the outer wall of the orbit, and supplies the lachrymal glands, the conjunctiva, and the skin near the external canthus. The *frontal nerve* enters through the same fissure, above the muscles, and internal to the lachrymal nerve. It passes forwards, and divides into the supra-orbital, whose exit from the orbit is the supra-orbital notch or foramen, and the supra-trochlear nerve, which passes over the superior oblique tendon and leaves the orbit internal to the supra-orbital nerve. The *nasal nerve* passes between the two heads of the external rectus, crosses the optic nerve beneath the superior rectus and superior oblique, and above the inferior rectus. It leaves the orbit through the anterior ethmoidal foramen. Its orbital branches are: (a) the long root to the ciliary ganglion, (b) the two long ciliary nerves which enter the globe on either side of the optic nerve (see p. 177), and (c) the infra-trochlear nerve, which passes beneath the superior oblique tendon, and leaves the orbit above the internal tarsal ligament, supplying the conjunctiva, the skin near the internal canthus, and the lachrymal sac. The *ciliary ganglion* lies in the posterior part of the orbit to the outer side of the optic nerve. Its three roots are derived from (1) the branch of the inferior division of the oculo-motor nerve to the inferior oblique muscle, (2) the nasal nerve, and (3) a filament from the cavernous plexus. The ganglion gives off the short ciliary nerves; they consist of two groups, superior and inferior, and enter the globe round the optic nerve (see p. 177).

The *superior maxillary division of the trigeminal nerve* enters the orbit as the *infra-orbital nerve*, through the sphenomaxillary fissure. It passes along the floor in the infra-orbital canal, and leaves by the infra-orbital foramen. Its orbital branches are chiefly periosteal. The *temporo-malar branch* of the superior maxillary

nerve enters through the same fissure, runs along its outer wall, and divides into temporal and malar branches, which leave through foramina in the malar bone. *Meckel's ganglion* also supplies orbital branches to the periosteum.

The *abducent nerve* enters the orbit through the sphenoidal fissure between the two heads of the external rectus, which it supplies on its ocular surface.

The *sympathetic filaments from the cavernous plexus* enter with the ophthalmic artery through the optic foramen, and pass to the ciliary ganglion, sending filaments to the non-striated muscle of the orbit.

The **orbital fat** fills up the intervals between the orbital contents, being at its maximum immediately behind the globe. It is divided into lobules by processes of fascia which enclose blood-vessels.

Cellulitis, or inflammation of the loose tissues of the orbit, may arise spontaneously, or may come on in the course of an attack of erysipelas.

In the milder cases the inflammation is localised; there are redness and œdema of the upper lid, and the conjunctiva is generally raised up by fluid either over its whole extent, or over a limited area. If the inflammation extends deeply into the orbit, the globe is rendered prominent, and, in most cases, the movements of the globe are painful.

In the severer forms the symptoms are all more marked. The inflammation may be ushered in with a rigor and a rise of temperature; there may be pain, swelling, and a dusky appearance of both lids; the globe is pushed forwards—*proptosis*; the conjunctiva is congested, and there may be considerable chemosis. The movements of the eye are limited, and there is consequent diplopia. From the diffuseness of the inflammation all the extra-ocular muscles, with the exception of the inferior oblique, are pressed upon and their action impaired, the eye is usually turned upwards and outwards in the direction of the action of the inferior oblique muscle, and attended with crossed diplopia, the false image being below the level of the true. Partial ptosis from impaired action of the levator palpebræ muscle is not uncommon. Visual acuteness may be much interfered with, and the globe may be involved in the inflammatory process. On digital examination between

the upper part of the globe and the orbital ridge, the tissues beneath are found to be tense, firm, and painful on pressure; in some cases semifluctuation can be felt. There is intense deep-seated throbbing pain.

The *causes* of this affection are various, and frequently obscure. It often follows erysipelas of the face; other causes are injury, septicæmia, inflammation of the lachrymal gland, periostitis.

Prognosis and treatment.—The milder forms are not dangerous; they usually subside by the use of fomentations every few hours, combined with dry warmth by means of cotton wool in the intervals, and general tonic treatment. The severer forms nearly always lead to suppuration, with the formation of *acute abscess of the orbit*. When this occurs, the pain becomes very severe, and the general symptoms more marked; there is considerable swelling of the lids with proptosis, and fluctuation may be detected. The absence of this latter sign must not cause any delay in making an incision if the other symptoms are sufficiently urgent; for if suppuration is present, and the pus finds no exit, it burrows among the ocular muscles, and may lead to their permanent destruction. The tissue of the optic nerve may also become involved, or the inflammation may spread through the orbital plate of the frontal bone to the meninges of the brain, or by the orbital veins to the cavernous sinus. A sharp scalpel should be passed either through the upper lid (both lids, if necessary) near the edge of the orbit, or through the conjunctiva, and then plunged deeply into the orbit, its point being directed away from the globe. Drainage must be provided for by tubes or gauze, and antiseptic fomentations applied, with frequent irrigation. Complications and sequelæ may arise—namely, panophthalmitis, necrosis of the bony walls, optic atrophy, and retraction of the lids.

Chronic abscess presents less marked symptoms, and is sometimes difficult to diagnose from a soft orbital tumour, which, owing to its elasticity, may appear to be semifluctuant. Pain may be slight, or altogether absent. There is usually some tenderness on pressure. The subconjunctival tissue is congested and swollen; there may be considerable proptosis

and lateral displacement. A history of some injury at a distant period will sometimes help in the diagnosis, and an exploratory incision into the semifluctuant region will often give exit to purulent matter.

Acute periostitis presents the same symptoms as acute abscess of the orbit; indeed, pus very rapidly forms beneath the periosteum, dissecting it from the bone, causing the death of the latter, and not unfrequently leading to the formation of an abscess in the anterior lobe of the brain, or to meningitis.

Treatment consists in making an early and free incision down to the bone. The inflammation is of a low erysipelatous type, and antiphlogistic measures are not well borne. A careful watch must be kept for the onset of cerebral symptoms.

Chronic periostitis is usually the result of syphilis, rheumatism, or scrofula. The pain is of a dull aching character, and is worse at night. When it affects the margin of the orbit there are œdema of the eyelid and tenderness on pressure. When deeply seated there is frequently paralysis of one or more of the ocular muscles, and sometimes prominence of the eyeball is present. The optic nerve and its sheath may be involved, retrobulbar optic neuritis resulting, with subsequent optic atrophy.

A gumma of the periosteum is not uncommon at the outer angle of the orbit. Diplopia from pressure on the globe is a frequent symptom. It readily yields under antisymphilitic treatment, a valuable aid in the diagnosis.

The *treatment* consists in giving full doses of iodide of potassium, combined or alternated with mercury, and counter-irritation is sometimes useful. In scrofulous cases it is nearly always the margin of the orbit that is affected; the treatment must then of course be directed to the general disease.

œdema of the orbital cellular tissue with exophthalmos occasionally occurs, and usually indicates deep-seated trouble in the circulation of the ophthalmic vein. This condition may be brought about by any pressure upon the vein in its passage through the sphenoidal fissure, such as often takes place in the case of periostitis, tumours of the optic nerve, and such like, and is then only a sign of embarrassed circulation; but when, in addition to these signs, we find the pupil widely

dilated, the globe quite immovable, and that cerebral symptoms are becoming manifest, the case is much more serious, and is indicative of *thrombosis of the cavernous sinus*.

Thrombosis of the Cavernous Sinus.—Though not in itself a disease of the orbit, yet so many orbital signs and symptoms occur as to justify its description under the title of this chapter.

It is a rare and most fatal affection, characterised by certain nervous and vascular disturbances within the orbit, which are attended by variable signs, subjective and objective. There is always a certain amount of *proptosis*, varying from slight prominence of the eyeball to a most pronounced exophthalmos; *paralysis* of one or more of the ocular muscles from pressure on their nerves while traversing the wall of the sinus; *frontal neuralgia* or *anæsthesia* along the course of the supra-orbital, supra-trochlear, and nasal nerves; possibly anæsthesia of the cornea, which may suffer from defective nutrition; *œdema* of the mastoid region, an important diagnostic sign; *œdema* of the conjunctiva, cellular tissue of the orbit or upper eyelid with distension of the frontal veins; the latter may not exist. Papillitis is not present in all cases, as one would expect if von Graefe's theory were true. The thrombosis of one sinus generally spreads by means of the sinus circularis to the cavernous sinus of the opposite side, with the development of similar signs on that side. Complete ophthalmoplegia externa may ultimately ensue. Pyrexia, vomiting, and rigors are, as a rule, present, together with cerebral symptoms. The cause is usually some injury, as fractured base, or meningitis secondary to otitis media, abscess of the orbit, &c. In some cases it is difficult to discover any cause. The prognosis is unfavourable; most cases terminate fatally.

Intra-orbital hæmorrhage is, as a rule, traumatic in origin (see p. 649), but in rare cases it may have a different etiology. In young children it is found in so-called scurvy rickets, when it is subperiosteal in nature, resembling the hæmorrhages occurring in other parts of the body. It occasionally takes place spontaneously during a paroxysm of whooping cough. Orbital hæmorrhages, again, may be due to rupture of a small aneurysm of a branch of the ophthalmic artery or of the main trunk itself, and also to a hæmorrhage into a vascular tumour.

The chief symptoms of this condition are the sudden onset, proptosis, impaired mobility of the eyeball, and the presence of blood beneath the conjunctiva and in the substance of the lids. The prognosis is good, but the cornea may slough owing to exposure unless great care is taken. Treatment should consist in the application of ice and a compress, rest in bed, a low but nourishing diet, and special attention to the general condition of the patient.

Emphysema, or the infiltration of air into the cellular tissues of the orbit, may be caused by rupture of the ethmoidal cells, or of the lachrymal sac. It may give rise to considerable proptosis, with swelling of the conjunctiva and eyelids. The swelling is increased when the patient makes a forcible expiration with the anterior nares closed, as in blowing the nose. It is characterised by a crackling sensation on digital examination, and can be reduced by firm pressure exercised over several days.

Distension of the frontal sinus—the result of pent-up secretion or pus—sometimes presents characters similar to those of orbital tumour. It is, as a rule, the result of an injury, such as a blow upon the forehead, although a long period usually elapses before the appearance of the swelling; not unfrequently it comes on in children after measles or whooping cough; it may be caused by extension of nasal catarrh, or may be secondary to neighbouring bone disease. The swelling first appears at the upper part of the inner angle of the orbit. At first it is hard, but after a time it may become soft and fluctuating, and a sensation of egg-shell crackling may be produced. The skin is freely movable over the tumour. It is usually slow in progress, but is liable at any time to take on acute suppuration. Before the bony wall has become absorbed, it may easily be mistaken for an exostosis, but it rises more gradually from the level of the adjacent bone, and, by firm pressure, some elastic yielding can usually be detected. In severe cases, the swelling is so extensive as to push the eyeball downwards and outwards. An acute osteo-myelitis may develop, the posterior wall of the sinus be involved, and a cerebral abscess formed in the frontal lobe.

Treatment consists in making an opening into the nose

to replace the normal exit afforded by the infundibulum. For this purpose, a free incision is made into the prominent part of the tumour, the mucus or pus evacuated, the interior of the sinus curetted, and the opening into the nose re-established. A drainage-tube is then passed through the hole and out at the nostril, and there retained for some time. The cavity must be syringed out frequently with an antiseptic lotion. Treated in this way, the swelling generally recedes, and the parts are sometimes restored to their normal dimensions. If osteo-myelitis is present, operative treatment must be very free, and all diseased bone removed.

Meningocele.—Owing to defective development of the upper and inner angle of the fore part of the bony orbit, the meninges may protrude and give rise to a hemispherical tumour in this region, which might possibly be mistaken for a dermoid growth, sebaceous cyst, or nœvus. It is usually a tense elastic swelling which pulsates synchronously with the heart-beat, and exists from birth. It can be reduced almost entirely by firm and steady pressure, and after its reduction the deficiency can be readily made out in the bony angle. There is an absence of half of the nasal process of the frontal bone and the adjacent part of the orbital ridge. A sebaceous cyst can readily be excluded in the diagnosis, for it is not congenital, not pulsatile, and cannot be reduced. Both a nœvus and dermoid cyst are congenital, liable to occur in this situation, and may really or apparently be reduced. The absence of any bony defect upon reducing the former and upon displacing the latter, besides the absence of pulsation, as well as other clinical features, are sufficient points to prevent any diagnostic error.

Exophthalmic goitre (Graves's disease; Basedow's disease) is the term applied to a group of symptoms of which the chief are: (1) paroxysmal cardiac palpitation, with throbbing of the vessels of the neck; (2) enlargement of the thyroid body; (3) prominence of the eyes; and (4) tremors, both regular and irregular, especially of the hands, and fine in character. It may, however, exist in the absence of proptosis on the one hand, or an enlarged thyroid on the other. It is usually ushered in by fits of caprice or irritability of temper; then come attacks of palpitation which are often very violent,

and are accompanied by a sense of suffocation, throbbing of the cervical vessels, and flushing of the face. Later, the throbbing of the neck becomes more or less permanent, and the thyroid gland is enlarged; this is from extreme vascularisation; the arteries carrying blood to the gland become larger, and the gland appears to be lifted *en masse* at each pulsation. The arteries within the gland become increased in size and number, and the veins convey arterial blood, so that the structure resembles a cirroid aneurysm; indeed, the elasticity and pulsation, together with the existence of blowing murmurs, have caused experienced observers to mistake this condition for aneurysm.

The enlargement usually begins, and is generally larger, on the right side. Hypertrophy of the connective tissue may or may not follow. Cystic bronchocele is a more rare accompaniment, and is probably a mere coincidence.

With the tachycardia, which persists on lying down, there may be dilatation of the heart, with a systolic murmur. The eyes begin to look prominent at the same time as, or a little earlier than, the thyroid enlargement; they have a shining appearance which, with the prominence, gives a peculiar frightened expression to the face. Lachrymation may be profuse. The proptosis is usually progressive, though stationary periods occur; it is generally equal on both sides. Strabismus is rare; occasionally, however, paralysis of one or more of the extra-ocular muscles, with the corresponding diplopia, is present. The retina is not appreciably altered, though pulsation of the retinal arteries may be seen. Vision is usually normal, as are also the pupils. Several cases of Graves's disease accompanied by increased tension, with all the signs of glaucoma, have been recorded. The sensibility of the cornea is diminished, and partly on this account, partly from exposure, corneal ulceration may arise, and progress until a panophthalmitis results.

Besides the proptosis, there are four other eye-signs which occur in this disease, all of which may be attributed to spasm of the muscle of Müller (see p. 4). They are as follows:

(a) *Von Graefe's lid-sign*.—The upper eyelid as a rule moves downwards *pari passu* with the eyeball, the relationship

between the lid and cornea remaining unaltered. In this disease, however, the lid does not move down so quickly as the eyeball, and even while moving down will often be suddenly and spasmodically raised, exposing a wide margin of sclera. Fig. 185 represents a photograph of a patient who suffered from this affection, and shows the exposed sclerotic



FIG. 185.—Bilateral von Graefe's Lid-sign.

coat in each eye. Fig. 186 is a case of unilateral von Graefe's lid-sign occurring in Graves's disease.

Von Graefe's lid-sign may precede the exophthalmos. Not only, however, may it be completely absent in Graves's disease, but it may be present when this disease is absent; hence it is not pathognomonic.



FIG. 186.—Unilateral von Graefe's Lid-sign.

(b) *Stellwag's sign* is also a peculiar feature in this complaint. There is a loss of the natural involuntary closure of the eyes (*blinking*). These blinking movements, if performed, are done consciously by voluntary effort; they are less frequent and may be in abeyance for some time, causing exposure of the cornea with desiccation of its epithelium.

(c) *Dalrymple's sign*, or undue widening of the palpebral aperture.—This is often very marked, and gives to the eyes

an appearance of staring. When one eye only is affected this sign is clearly observed. It may give rise to *apparent proptosis* when no real proptosis exists.

(d) Besides these phenomena, there is an *increase in depth of the orbito-palpebral fold or sulcus*, due undoubtedly to the same cause.

It must be carefully borne in mind that the eyes themselves are not enlarged, but are simply pushed forwards by the vascular distension of the fatty connective tissue at the back of the orbit. There is a venous stasis of this tissue, causing it to become turgid like erectile tissue—a simile which Graves himself used. The eyeballs usually recede *post mortem*. True hypertrophy of the retrobulbar tissue is, however, sometimes found, but is probably secondary to the hyperæmia.

Sleeplessness is a common symptom, especially early in the case. A more or less permanent *febrile condition* is sometimes observed (Frissier, Basedow). The *appetite* may fail, or may be greater than in health. *Vomiting* is common, and the patient grows thin even when the appetite is good. *Diarrhœa* is common, and usually alternates with constipation. In women there is generally *amenorrhœa*, usually accompanied by profuse *leucorrhœa*. Stokes thought the whole disease due to *anæmia*, but cases have occurred without any *anæmia* (Frissier).

Etiology and pathology.—The disease is far commoner among women, and Trousseau states that out of fifty cases collected by Withuisen, only eight occurred in men. The age is most commonly from twenty to twenty-five, or a few years earlier, but the disease is rare in advanced life. Occasionally it is unilateral, and if so is generally right-sided.

The patients are usually nervous subjects, and often have a history of hereditary nervous disease. Several cases have been traced to some shock, such as fright or grief, but, as a rule, no cause can be assigned. Trousseau and many others ascribe the disease to derangement of the cervical sympathetic nerves and ganglia, especially the inferior cervical ganglia; hence paresis of the vaso-motor system, and consequent dilatation of the vessels. In various autopsies the above ganglia

have been found diseased, showing hypertrophy of the interstitial connective tissue, and atrophy of the nervous elements. But other most careful observers (Ranvier, Wilks, Déjérine, Cheadle) have failed to find any abnormal appearances in the sympathetic. Four signs have been attributed to a spasmodic action of the muscle of Müller for the following reasons: in paralysis of that muscle there is slight ptosis or narrowing of the palpebral fissure with loss of the natural orbito-palpebral fold, though upon looking upwards the sulcus appears again from the action of the levator palpebræ muscle, which has a cutaneous attachment. It is, I presume, through the connection that the muscle of Müller has with the tendon of the levator palpebræ that this fold or sulcus exists in health in the primary position without voluntary effort. If *cocaine* be instilled into the palpebral sac all the above signs can be produced. We have sufficient proof that cocaine stimulates unstriped muscular tissue by its hæmostatic action, by its bleaching an injected conjunctiva, and above all by its power of dilating the pupil. This dilatation of the pupil is caused partly by the direct stimulation of the dilator pupillæ, and partly by the contraction of the unstriped muscular fibres in the walls of the iridic vessels, which differ in one respect from those in other vessels, viz. in running mainly parallel to the long axis of the vessel. From these facts I conclude that cocaine stimulates the muscle of Müller and not the levator palpebræ muscle, for there is no evidence that it stimulates voluntary muscular tissue. I feel equally convinced that this Müllerian muscle is in a state of tonic contraction in Graves's disease, since the signs are identical; though Sharkey considers that these signs are due to a weakness of the orbicularis palpebrarum muscle, I have not noticed any weakness in this or any of the other facial muscles. It is clear, therefore, that this disease cannot be due to a lesion of the cervical sympathetic cord, for there exist evidences of both irritation and destruction at the same time. If the irritation were premonitory to paralysis, the spasm of Müller's muscle would be a transient sign and associated with temporary contraction of the pupil, but such is not the case. Cheadle, in an interesting case described in the 'St. George's Hospital Reports,' found

considerable capillary dilatation in the medulla oblongata and upper part of the spinal cord, but without atrophy or cellular lesions, showing thus simply *increased vascularisation*. No lesions were found elsewhere, either in the viscera or in the cervical sympathetic. The pneumogastric nerve is certainly implicated, as shown by the disturbances of the digestive tract, and the palpitation of the heart. The singular nervous sensibility, which is so early and constant a symptom, and which in some cases has even gone on to mania after the cure of both the exophthalmos and the goitre, would seem to point to the brain itself as the initial seat of the disease. Sattler considers that the lesion is central, involving the vaso-motor centres presiding over the thyroid vaso-motor nerves and the orbital vaso-motor nerves respectively, and also the cardio-inhibitory centre for the vagus. A toxic theory has found favour with some, excessive secretion by the thyroid gland being held to be the main factor in the causation of the disease.

Prognosis.—It is uncommon for a complete cure to occur, though many cases are greatly benefited by treatment. As a rule, progress is very slow. Occasionally the proptosis increases rapidly, both eyes being lost. Exhaustion may end in death.

Treatment.—Special attention must be paid to the general health. The avoidance of mental emotion is very important. Digitalis is lauded by Trousseau, together with the application of ice to the præcordium and the thyroid body. Bromide of potassium is useful, and so are opium and chloral. Belladonna, which theoretically would be bad, is practically found to be of great benefit. Veratrum viride carefully given is much praised by Aran and Sée, since it makes the pulse slower without increasing the arterial tension, thus differing from digitalis. Other drugs that have been used are strophanthus, quinine, potassium iodide, aconite, ergot, large doses of phosphate of sodium, and extract of the thymus gland.

Iron has been found harmful. The galvanic and faradic currents applied to the neck have been found to be beneficial in many cases.

With regard to operative treatment, results are on the whole unsatisfactory. The thyroid gland has been totally or partially removed in a few cases; the former operation is quite

unjustifiable. Others, who hold to the sympathetic origin of the disease, have excised a part of the cervical sympathetic nerve, with or without the superior cervical ganglion.

Threatened asphyxia must be met by excision of the thyroid isthmus, or by a timely tracheotomy.

Ulceration of the cornea indicates a tarsorrhaphy, but if at all extensive, usually necessitates enucleation. It must be remembered that the subjects of exophthalmic goitre take anæsthetics badly, and are very liable to syncope after operative measures.

Tumours of all kinds are found in the orbit; they may originate within the tissues of the cavity, they may commence within the eye and thence extend to the orbit, or they may invade that cavity from surrounding parts, as the nose, the palate, the antrum, the skull, or the temporal fossa. Orbital tumours may be non-malignant and of slow growth, as the cystic, the fibrous, and the fatty kinds; they may be malignant and more or less rapid in progress, as the sarcomata and carcinomata, or they may be pulsating, as the vascular tumours.

Symptoms.—The presence of a tumour of any notable magnitude always gives rise to protrusion of the globe (proptosis). When the tumour is deeply seated, and at the apex of the orbit, this is usually one of the first signs of its existence; when situated at one side of the orbit it, as a rule, causes lateral as well as forward displacement. In proportion to the increase of the tumour, the globe becomes displaced, until in severe cases it is protruded beyond the palpebral aperture. Though the tumour may not be sufficiently anterior to be palpated, in attempting to press back the eyeball a sense of increased resistance may be felt. Functional troubles are, also, always present, and will vary according to the position and size of the swelling; when the cranial nerves are pressed upon, pain will be severe, and the movements of the globe impeded; should the optic nerve be involved, its function will be interfered with, and the vision partly or entirely destroyed, optic neuritis or atrophy being visible with the ophthalmoscope; with lateral deviation, where vision remains, diplopia is always produced. The mobility of the eyeball is usually more or less impaired. Pressure upon the ophthalmic vein is likely to set

up œdema of the orbital tissues, and distension of the conjunctival and retinal vessels, with perhaps retinal hæmorrhages. Proptosis is not always easy to make out; it may be mistaken for enlargement of the globe such as is sometimes found in progressive myopia and secondary glaucoma. In such cases, if the upper lids are elevated by the surgeon standing behind the patient, who is told to look downwards, the antero-posterior elongation of the affected globe will be at once detected. Tumours in the upper part of the orbit often cause ptosis. Corneal anæsthesia and various forms of keratitis may be present.

After a time, the tumour becomes apparent at some part of the margin of the orbit, and can then be examined by palpation, by auscultation, and, if necessary, by exploratory punctures. Whenever an orbital tumour is found to exist, the condition of all surrounding regions, as the mouth, pharynx, and nasal cavities, should be carefully examined.

Neuroma, Lipoma, and Fibroma are extremely rare in the orbit; a few cases, however, are recorded.

Cysts occurring in the orbit are not uncommon; they are usually either *dermoid* (see p. 14) or *hydatid*.

Hydatid cysts, the cystic form of the *tænia echinococcus*, within the orbit are also very rare, but may be a cause of proptosis; the signs in such a case would simulate retrobulbar new-growths, as a sarcoma of the optic nerve, &c. They can only be correctly diagnosed by finding upon aspiration a clear non-albuminous neutral saline fluid containing the characteristic hooklets. It is most advisable to make an exploratory incision and aspirate a doubtful orbital tumour before removing the orbital contents on the assumption that it is a sarcoma. Suppuration may occur in these orbital cysts as in those in other parts of the body. The *treatment* consists in opening and evacuating the cyst and washing out the cavity with a strong antiseptic solution.

Cysticercus cellulosæ, the cystic form of the *tænia solium*, within the orbit is even more rare than the preceding.

Exostoses of the orbit are similar to those occurring in other parts of the body. They are usually of the ivory variety, attached to the frontal or ethmoidal bone, and

adherent by a broad base ; in such cases it is impossible to remove them. Occasionally, however, they are pedunculated, and are then amenable to treatment. The periosteum should be stripped off, and small holes drilled into the tumour with the dental or electro-motor drill, followed, if necessary, by the chisel. Such operations are, however, not altogether free from risk when, as is usually the case, the growth is attached to the roof of the orbit ; for a portion of the latter may easily be torn away, and a fatal meningitis set up. Occasionally spontaneous separation by necrosis of the pedicle takes place.

More rarely exostoses are met with which contain large cystic cavities communicating with each other ; this variety sometimes attains enormous dimensions.

Exostoses of the orbit are slow in development, and painless in progress, producing in succession all the symptoms that have been above enumerated as characteristic of intra-orbital tumour.

The diagnosis of orbital exostoses is often somewhat difficult, as they closely simulate distension of the frontal (see p. 611) or ethmoidal cells.

Sarcoma is the most frequent of the new-growths affecting the orbit. It may occur by extension from the choroid (p. 220), it may originate in either the periosteum or bone, or it may first appear in the cellular or muscular tissue of the orbit. Its rate of progress is very variable ; when it develops rapidly the tumour presents but little pigmentation, whilst the slow-growing sarcomata are usually dark in colour, and are sometimes quite black. They are very frequently found to have undergone myxomatous changes.

The *treatment* consists in the early and complete removal of the diseased tissues. When the tumour is small, circumscribed, and near the surface, it can occasionally be removed without molesting the globe of the eye. To facilitate this, the palpebral opening may be enlarged by dividing the lids at the outer canthus, and then dissecting in the direction of the tumour ; which, when exposed, may be seized with vulsellum forceps and cleared from its surroundings by means of a steel director, or by strong curved blunt-ended scissors, and then cut or torn away.

Sometimes a small orbital tumour can be removed by an incision through the skin at the margin of the orbit without interfering with the conjunctival sac.

When the tumour or new-growth is extensive, and involves the tissues of the orbit, or has recurred after removal, it is necessary to take away the globe and the whole of the orbital contents. To effect this, the external canthus must be divided up to the edge of the orbit, the conjunctiva separated by incision through the whole extent of the upper and lower culs-de-sac. The eyelids are then seized with forceps or retractors, and drawn upwards and downwards by an assistant. The globe can now be enucleated before taking out the tumour, or the whole mass, including the eye, can be seized with vulsellum forceps and pulled forwards, while it is detached from the walls of the orbit with strong curved blunt-ended scissors. Having thus removed the greater part of the tumour, careful digital examination must be made, and any further portions of tissue which appear to be diseased removed. Hæmorrhage is usually copious, but generally ceases after pressure with sponges or pledgets of cotton wool soaked in extract of the suprarenal body. If it cannot be controlled by these means, a button-shaped thermal cautery at a dull-red heat may be used. It is often advisable to remove the eyelids; if left, a troublesome entropion is liable to be produced.

Carcinoma occurs in the orbit either primarily or by extension from surrounding parts. The primary form is very rare.

The *treatment* consists in the complete removal of the diseased tissues.

Pulsating Exophthalmos.—Cases are occasionally met with in which the globe becomes protruded at the same time that a soft pulsating swelling, with aneurysmal bruit and thrill, appears usually at the upper and inner angle of the orbit. The pulsation is by no means always visible. The conjunctiva is frequently in a chemosed condition, and the cornea very prone to ulcerate and become anæsthetic. If the fundus details are visible, the vessels will be seen to be greatly distended, and papillitis present. Compression of the common carotid often diminishes the pulsation and the sounds.

The history usually given is either that the patient has

suddenly heard a loud snap, and that this has been succeeded by an intermittent buzzing or blowing noise, and soon afterwards by the pulsating swelling; or that the symptoms have come on very shortly after a severe injury to the head. In the majority of these cases, symptoms of fracture of the base have been present.

In a third class, a perforating wound either in the orbit or the roof of the mouth has been the immediate cause.

In the earlier cases the symptoms were supposed to be due to intra-orbital aneurysm—an opinion which was strengthened by the discovery of such aneurysms in two cases by Guthrie and Carron du Villards. Later autopsies have proved that, as a rule, the pulsating swelling in the orbit is formed, not by the ophthalmic artery, but by the varicose and distended ophthalmic vein, and that this distension can be traced back to the cavernous sinus, between which and the internal carotid artery a communication often exists. This aneurysmal varix may be brought about by the giving way of an atheromatous patch,¹ by the rupture of an aneurysm in the sinus,² by a fracture of the base passing across the sinus,³ or by a wound. Thus in one of Nélaton's cases,⁴ the rib of an umbrella thrust into the right orbit passed through the body of the sphenoid and wounded the left carotid artery as it lay in the sinus; the injury was shortly followed by pulsating exophthalmos on the left side.

In Schaefer's case the artery was wounded by a pistol-shot fired into the mouth.

In a few cases both orbits have been affected; this occurred in a case published by W. Adams Frost:⁵ the patient at the time of observation was thirty-eight years old. When ten years of age he had been run over by a timber-wagon. He had symptoms of fracture of the base, and the characteristic symptoms of pulsating exophthalmos appeared in the left orbit and had persisted ever since; shortly before he came

¹ Hirschfeld, *Gaz. des Hôpit.* 1859, p. 57.

² Baron, *Med. Chir. Trans.* xlviii.; Nunneley, *ibid.* xlii.

³ Nélaton, *Delens de la Communication de la Car. Int. et du Sinus Cav.* Paris, 1870.

⁴ *Ibid.*, *Delens, loc. cit.*

⁵ *Trans. Ophth. Soc.* vol. iii. p. 9.

under observation a small pulsating swelling appeared in the right orbit.

An idiopathic variety is perhaps due to aneurysm of the ophthalmic or internal carotid artery, or to thrombosis of the cavernous sinus.

In a few cases the symptoms have been due to a malignant tumour in the orbit.

A congenital form of pulsating exophthalmos occasionally occurs. This is probably due to a pulsating nævus, and especially indicates electrolysis as the method of treatment.

Treatment and prognosis.—Ligature of the common carotid artery has been extensively employed, and with a fair amount of success, though cases have been reported in which the exophthalmos has recurred after an apparent cure; other measures, such as rest, low diet, application of ice, application of digital pressure to the common carotid on the same side, galvano-puncture, electrolysis, and the injection of styptics, have also been successful in some cases. The affection, however, tends, after having reached a certain stage, to become stationary, and not unfrequently undergoes spontaneous cure; so that unless the noise in the head were distressing, or the increase in the size of the swelling rendered its rupture probable, a prudent surgeon would not adopt such of the above modes of treatment as are fraught with danger to life.

Temporary Exophthalmos.—This peculiar and rare condition occurs only on stooping or on pressing the jugular vein. In the erect position exophthalmos may be present. There is sometimes a history of injury. The probable explanation is that there is an orbital angioma, or that the retro-ocular veins are varicose.

Erectile or Cavernous Tumours.—These growths, whose structure resembles very much that of the corpora cavernosa, seem to be more frequent in the orbit than elsewhere. They are slow-growing, but tend to mould themselves to the parts with which they come in contact, so that their removal *en masse* without injury to the optic nerve and muscles is generally impossible, while their great vascularity renders a partial operation troublesome and dangerous.

CHAPTER XVII.

INJURIES OF THE EYEBALL AND ITS APPENDAGES.

INJURIES OF THE EYELIDS.

INJURIES of the eyelids are similar in nature to those elsewhere, viz. contused, punctured, incised, and lacerated.

Simple contusions are very common, and may vary from slight redness to severe cutaneous and subcutaneous ecchymoses ('black-eye'). They are not unfrequently accompanied by serious lesions of the eyeball and orbit. Hæmorrhage into the eyelids may be a sign of fracture through the anterior fossa of the base of the skull. The absorption of the ecchymosis in 'black-eye' is often hastened by evaporating lotions. Cold applied at the time of the blow, as the popular remedy of raw steak, will arrest further hæmorrhage and so limit the extravasation.

Punctured wounds are caused by stings of insects, lead pellets, and sharp-pointed instruments. Stings from gnats, wasps, bees, hornets, &c., cause smarting, redness, and œdema, sometimes to an alarming extent, but are rarely serious; the puncture should be touched with liquor ammoniæ, and fomentations applied. Gunshot accidents are a cause of grave anxiety. It is necessary to ascertain the direction of the pellet or pellets, and the state of the eyeball. If the shot can be felt with a probe and is not deep in the orbit, its removal may be effected by enlarging the wound parallel to the course of the fibres of the orbicularis palpebrarum and removing it with forceps. Injuries to the globe will be considered later. It is a dangerous practice to undo knots in bootlaces, string, &c., with a sharp-pointed tool, as a pair of scissors, an awl, and the like; the instrument often slips, and

the liability to penetrate the eyeball, directly, or indirectly through the lid, is very great.

Wounds, when *incised* or *lacerated*, should, if uncomplicated, be cleansed with a suitable antiseptic lotion, and have their edges neatly adapted with sutures. Wounds parallel to the muscular fibres do not gape at all, but those at right angles to them do so considerably; and if the margin of the lid is divided throughout its thickness, an imperfect union will result in a permanent traumatic coloboma. As complications may be mentioned (*a*) those due to infection, as erysipelas, cellulitis, abscess, &c., and (*b*) those due to anatomical accidents; as division of the levator palpebræ tendon with consequent ptosis, penetrating injury of the eyeball, wounds of the orbital wall, especially the sinuses, with resulting emphysema, division of the canaliculi, ectropion, symblepharon, &c., and, as a very rare sequela, an implantation cyst.¹ If the tendon of the levator is divided, an attempt should be made to suture it; in these cases, however, the damage to the eyeball is usually so great as to necessitate its removal, and the difficulty of securing the tendon increased. Simple wounds heal rapidly. They should be cleansed, united with sutures, and protected with an antiseptic dressing. In treating a vertical incision through the eyelid, a suture near its margin must be inserted through its whole thickness, so as to bring the cut surfaces in close apposition without tension; two or more cutaneous sutures will be required. If a triangular piece of the lid has been lost, tenotomy of the tendo oculi may materially assist the closure of the gap. *Emphysema*, or the diffusion of air beneath the skin of the eyelids, or even beneath the ocular conjunctiva, is caused by a fracture involving the lachrymal sac, the nasal fossæ, or one or other of the different sinuses surrounding the orbit, followed by the escape of air into the cellular tissue. With such a solution of continuity, violent blowing of the nose will bring it about; in other words, it results from increased pressure in the nasal cavities or their sinuses. The characteristic crepitation on palpation is easy of recognition and absolutely diagnostic. Though an alarming

¹ Anderson Critchett and Griffith, *Ophth. Soc. Trans.* vol. xvii. p. 242.

symptom, it may be regarded lightly, as the air is quickly absorbed without inflammation.

Burns of every degree and description may occur in this region, from mere hyperæmia to complete charring of the eyelids and eyeballs. They may be caused by falls on the fire: especially is this the case with young children, epileptics, and those who suffer from syncopal attacks; by explosions of gas or gunpowder, especially fireworks; by boiling liquids, as water, oils, fats, metals, &c.; by corrosive liquids, as the strong acids and alkalies, sometimes maliciously thrown into the face for the purpose of destroying the features; by quicklime, and in various other manners.

As with injuries so with burns, it is necessary in the first place to discover the extent of the mischief. As the pain is, as a rule, most severe, it is often advisable to administer a general anæsthetic before making an examination. If it is possible to do so with cocaine, the greatest gentleness is required. The eyelids swell early; and with bullæ, some distended, others ruptured, combined with blepharospasm, it is no easy matter to obtain a view of the eye; to evert the lids is next to an impossibility. Where lime or metal has entered the eye, this must be done so as to remove all particles and cleanse the palpebral sac. Castor oil and cocaine should be instilled, and after a few minutes the palpebral sac may be irrigated with warm boric acid lotion, the lids everted, and all fragments of lime or metal carefully removed with a corneal spud. Where destruction of tissue has occurred, the sloughs must be removed as they separate, and ulcers stimulated to heal rapidly. Any disfigurement from ectropion, or from adhesions of the raw edges of the eyelids together (ankyloblepharon), or between the eyelids and eyeball (symblepharon), must be prevented by passing a probe between the ulcerated surfaces each day. Burns from acids should be bathed with a solution of bicarbonate of soda, those from alkalies with weak acetic acid or vinegar. In gunpowder explosions the face, eyelids, and eyeball are peppered and ingrained with carbon particles, and the disfigurement, if they are left, is great. The sooner the pigment is removed the better. Under an anæsthetic each spot should be scooped out

with a very small curette, and practically no scars remain. The pigment granules in the ocular conjunctiva, if few, may be snipped out with curved scissors, and those on the cornea removed with a spud.

Ankyloblepharon signifies the adhesion of the ciliary margins of the eyelids together. It may be congenital or acquired, complete or partial. It often accompanies and is produced by the same cause as symblepharon. The adhesion is rarely so complete as to involve the entire edges of the lids—it usually occupies only their outer half; even in the most complete cases a small opening, as a rule, exists near the inner canthus, through which the tears and mucus can escape. The *treatment* consists in dividing the cicatricial structures which hold the lids together. To do this, a grooved director should first be passed behind the lids, and the incision made with a small scalpel.

Symblepharon is the abnormal adhesion of the eyelids to the globe. It is usually caused by burns or injuries, but occasionally follows trachoma, pemphigus, and diphtheritic conjunctivitis. Symblepharon may be *partial*, consisting of one or more bands of cicatricial tissue extending from the conjunctiva of the lid to that of the globe, and thus forming a bridge of tissue beneath which a probe can be passed; or it may be *complete*—that is, the entire surface of the affected portion of the lids becomes united to the globe. The lower lid is most commonly adherent; in severe cases this becomes united to the cornea, thus producing great deformity, limitation of the upward and lateral movements of the globe, and partial or total loss of vision (see fig. 187).

Treatment.—In the simpler forms of partial symblepharon, where only a band of cicatricial tissue extends from the palpebral to the ocular conjunctiva, and where a probe can be passed beneath, it is usually sufficient to snip away the adhesion close to



FIG. 187.—Symblepharon. (After Anderson Critchett.)

both surfaces with scissors, and to keep the raw surfaces from uniting by separating them with a probe every day. When more extensive adhesions exist, we must have recourse to other procedures.

1. **Teale's operation** consists in the dissection of the adherent lid from the globe, so that the latter can move freely in all directions. This done, the neighbouring healthy conjunctiva is utilised, by dissection and stretching, so as to form flaps to cover the ocular and, if possible, the palpebral surfaces. Numerous fine silk sutures are used to draw the edges of the new flaps together. Various modifications of this operation are performed by different surgeons.

2. In more severe cases, where there is not sufficient ocular conjunctiva to admit of its being utilised, the conjunctiva of another eye or from that of a rabbit, or the mucous membrane from the patient's lip, may be transplanted.

INJURIES OF THE LACHRYMAL APPARATUS.

The lachrymal gland may be involved in a *burn* of the eyelid, and, as a consequence, more or less completely destroyed. Occasionally *acute dacryo-adenitis* (see p. 45) seems to be set up by an injury. Division of some wall of the lachrymal ducts may produce a retention cyst, or *dacryops* (see p. 48). As an extremely rare result of trauma may be mentioned *dislocation* of the gland, which usually necessitates its removal.

The Puncta Lachrymalia.—*Displacement* of a punctum is usually the first sign of ectropion, and has, consequently, the same etiology. *Stenosis* may be produced by a burn. Foreign bodies, as a hair, may be caught in a punctum.

The Canaliculi.—Burns may cause *stenosis* (see p. 50) of one or both canaliculi. As a result of a wound in the neighbourhood of the internal canthus, a canaliculus may be completely *divided*. Attempts should be made to find the divided ends, and the canaliculus should be slit up and kept open.

The lachrymal sac may be injured by a fracture of the lachrymal bone.

The Nasal Duct.—One of the causes of *stricture* (see p. 52) is an injury to the nasal bones.

INJURIES OF THE CONJUNCTIVA.

Burns and chemical injuries are of frequent occurrence. Hot water, quicklime, cigar-ashes, and molten metal are frequent causes of this trouble. The action of these agents is very similar to that of strong caustic: the mucous membrane is partly or entirely destroyed at the part affected, and is likely to be followed by cicatrization. This fact has to be borne in mind during the treatment of the case, and the prognosis will be much influenced (1) by the extent to which the cornea has been involved in the accident, and (2) by the extent of the conjunctiva that has been doomed to destruction. If both palpebral and ocular conjunctivæ are injured, there is great danger of symblepharon.

Treatment.—First, all traces of the offending substance must be removed from the palpebral sac, and the latter carefully washed. Strong chemicals should be neutralised by appropriate solutions. Secondly, cold compresses should be applied, and atropine and cocaine drops instilled. At each daily dressing the eyelids should be well separated from the globe in order to prevent their growing together, and a lubricating emollient, as olive or castor oil, dropped into the palpebral sac. Should the injury have extended as far as the fornix, it will be found impossible to prevent union of this kind. (See Symblepharon, p. 627.)

Wounds of the conjunctiva may be mere abrasions caused by some foreign body, or they may be extensive, and are then either incised or lacerated. They are frequently accompanied by some subconjunctival hæmorrhage. After well irrigating the palpebral fissure with an antiseptic lotion, the edges of the wound, if extensive, should be trimmed if necessary, and brought together by fine sutures.

Foreign Bodies.—Small foreign bodies, as dust on a windy day, bits of cinder on the railway, or particles of steel, often enter the palpebral sac. As a rule, they cause considerable irritation, lachrymation, and photophobia; this is much intensified if the particle be situated on the inner surface of the upper lid and rub against the cornea. This is a very common situation for foreign bodies. They are easily seen and removed by everting the upper lid.

Sometimes sharp particles become fixed in the cornea or in the ocular conjunctiva, and occasionally they find their way into the upper cul-de-sac.

In searching the upper cul-de-sac the patient should look well downwards, and whilst the everted upper lid is pulled upwards the globe should be pressed upon by the non-everted lower lid. This causes the upper fornix to bulge forwards.

NON-PENETRATING INJURIES OF THE CORNEA.

Non-penetrating injuries of the cornea may be considered under three headings—Burns and scalds, Superficial wounds, Presence of foreign bodies.

Burns and scalds are produced by quicklime, mineral acids, caustics, boiling water, strong ammonia, fusing metals, gunpowder, curling-tongs, and the like. The action of quicklime upon the cornea is very destructive, more so than the appearance of the cornea immediately after the accident would lead us to imagine. The surrounding limbus conjunctivæ is swollen and yellowish-white from solid œdema, the ocular conjunctiva is blanched and spotted here and there with petechial hæmorrhages. When only the superficial portion of the corneal tissue is cloudy, the deeper parts remaining transparent, we may hope for some preservation of vision; but when there is a diffused and deep grey appearance, the prognosis is very unfavourable.

Treatment must be immediate. Both the eyelids should be everted and thoroughly cleansed. If the injury has been produced by quicklime, a strong solution of sugar should be used for irrigation, the lime being dissolved in the soluble salt of calcium which is formed. Acids must be neutralised with bicarbonate of soda solution, and alkalies with weak citric or acetic acid. When all of the offending substance has been removed from the conjunctival sac, a drop of $\frac{1}{2}$ per cent. solution of atropine should be placed in the eye, and a slight compress applied. Should the conjunctiva and neighbouring parts become much inflamed, soothing lotions and atropine and cocaine drops must be employed. Sloughs as they form must be removed. The eyelids must be opened

daily, and precautions taken to prevent adhesions between the globe and the lids (see Symblepharon), and also between the margins of the lids themselves (see Ankyloblepharon, p. 627). There is frequently considerable shock to the general system, and stimulants are often necessary.

Superficial wounds may consist of a simple abrasion, or a scratch, with or without contusion. These injuries usually heal without trouble; they simply require that the eye should be thoroughly cleansed, and that a few drops of $\frac{1}{2}$ per cent. solution of atropine and a light compress should be applied. If there is any doubt as to abrasion of the cornea, a drop of solution of fluorescein (F. 18) will at once decide the question by rendering the abraded surface green and opaque. A nebula or leucoma may remain at the seat of injury, and the patient should be prepared for this defect, which may interfere with the vision of that eye. When there is any persisting purulent affection of the injured eye, such as dacryo-cystitis, or granular conjunctivitis, the cornea is less able to recover from the traumatism. The wound may become inflamed, and suppuration with hypopyon supervene. (See Suppurative Keratitis.)

Foreign bodies in the cornea are of frequent occurrence, and of great variety. Those most commonly met with are small bits of metal, coal-dust, and sand.

The presence of a foreign body in the corneal tissue is marked by immediate pain, photophobia, and lachrymation; the pain is most intense when the substance is so situated as to be rubbed against and pressed upon by the eyelid. If not quickly removed, local keratitis is set up. The presence of a foreign body is sometimes difficult to recognise, especially when it is very small; but by careful examination with oblique focal illumination (p. 110) and a corneal magnifier it can always be detected.

Immediate removal is in all cases imperative. The difficulty of this will depend upon the depth to which the particle has become embedded in the tissue. Cocaine should first be instilled to produce anæsthesia of the cornea and flaccidity of the eyelids. For ordinary cases, in which it is situated on a level with the surface, the surgeon stands behind the patient, who is seated in a good light, with his head thrown back and

protected by a towel, so that it can be steadied against the surgeon's chest; the eyelids are now separated by the fingers of the left hand and the globe held in position by firm pressure of the same fingers against the ocular conjunctiva. The patient is directed to look in such a direction as may bring the foreign body most clearly into view, and to fix his vision in that direction as much as possible. A small spud (fig. 188) is now used; this should be passed fairly beneath the embedded particle, which can then be elevated and removed. A cataract needle may sometimes be advantageously substituted for the spud.

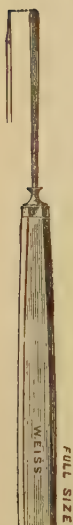


FIG. 188.
Corneal
Spud.

When the foreign body is deeply embedded in the cornea, so that it touches or even perforates Descemet's membrane, it may be impossible to remove it by the above method; in this case a broad needle should be passed through the cornea into the anterior chamber, in such a way that the flat portion of the needle can be passed behind the part where the foreign body is embedded. A little pressure is here made, and the point of a Beer's cataract knife or a keratome can now be used to cut down to the particle, and remove it without fear of its falling into the anterior chamber. This done, the broad needle is withdrawn. Great care must be taken not to wound the crystalline lens. A drop of atropine solution is used, and the eye closed by a light compress for a few days.

When a foreign body is allowed to remain in the cornea, it establishes local keratitis, which may be very severe and extend to the whole cornea; the surrounding tissue becomes hazy and rather swollen, and the particle sooner or later becomes loose and detached. The resulting opacity in this case is much greater than it would have been had the particle been removed at once; and in the case of some metals there is often a considerable stain left from deposit of the oxide.

In explosions from *gunpowder*, numerous particles of carbon may enter the eyes, damaging the cornea and conjunctiva. The eyelids and skin of the face also suffer severely,

and if the carbon be not removed, permanent disfigurement is the result. The cornea should be dealt with first, and each granule removed by means of a corneal spud or cataract needle. Then the ocular conjunctiva should receive attention, small pieces of which it may be necessary to remove. A very small curette is the best instrument for dealing with the skin. Great care is necessary in cleansing the skin and eye, otherwise erysipelas is very prone to ensue.

NON-PENETRATING INJURIES OF THE SCLERA.

Superficial wounds are of but slight importance, and usually heal without trouble.

Contusions of the sclera without rupture are only of importance according as they affect the internal structures of the eye. It is not uncommon to find dislocation of the lens, partial detachment of the iris, rupture of the choroid, and hæmorrhage into the anterior chamber or vitreous resulting from a severe blow upon the eye, which may not have caused rupture of the sclera.

Foreign bodies lodged in the sclera are rare, since they either penetrate the globe or rebound.

NON-PENETRATING INJURIES OF THE IRIS AND CILIARY BODY.

Severe contusion of the eyeball without perforation may injure the iris in several ways.

Rupture of a blood-vessel may be the result of the blow. This is characterised by the presence of blood in the anterior chamber (hyphæma). The eye must be carefully examined for other lesions of the iris and of other structures, but it may be necessary to wait until the blood has been absorbed before this can be done. Should other lesions be absent, the prognosis is good, and the blood will become absorbed in a few days. The eye should be shaded, and kept at rest by means of atropine; no reading must be attempted. Very rarely iritis supervenes.

Traumatic mydriasis is probably always due to some laceration of the iris, and is consequently usually accom-

panied by hyphæma. The laceration is radial, and may implicate only the pupillary border, being often so minute as to require high magnification before it can be seen. Paralysis of the sphincter pupillæ—*iridoplegia*—results, and with it may be combined paralysis of accommodation—*cycloplegia*. Iridoplegia may occur without cycloplegia, but traumatic ciliary paralysis alone is rare. The treatment consists in rest in bed, and the application of cold to the eye. Atropine should not be used unless iritis is threatened, which is very uncommon. The prognosis must be guarded, as the condition is often permanent.

Irido-dialysis, or detachment of the iris from the ciliary body, is usually only partial. The ciliary border of the iris is its weakest part, receiving little or no support behind from the lens, and consequently is subjected to the greatest strain when the cornea is flattened by a blow. Hyphæma is always present, and if severe may hide the detachment. The *circulus arteriosus iridis major* is probably ruptured together with Schlemm's canal. The appearance is that of two irregular pupils. Ophthalmoscopically, a pupillary reflex may be obtained through the gap, while the ciliary processes and the suspensory ligament of the lens, unless it has been ruptured by the blow, may be seen. The patient may complain of diplopia, which will be found to be monocular. The prognosis is bad, as the condition is usually permanent. A few cases of spontaneous cure have, however, been recorded. The treatment should be iced compresses and the local application of atropine, which will help to approximate the edges of the gap.

Traumatic aniridia, or *irideremia*, is the name given to total irido-dialysis. The totally detached iris floats forwards and sinks to the lower part of the anterior chamber.

Retroflexion and **anteflexion** are very rare dislocations of the iris, and are usually only partial. In retroflexion, a part of the iris is doubled back so as to cover the ciliary processes, the suspensory ligament being ruptured with dislocation of the lens. The result simulates in appearance a coloboma, but the ciliary processes cannot be seen, since they are hidden by the retroflexed iris. In anteflexion, the ciliary border of the iris is not only detached from the ciliary body, as in irido-

dialysis, but is doubled forwards over the rest of the iris, the posterior pigmented layer coming into view.

NON-PENETRATING INJURIES OF THE CHOROID.

Rupture of the choroid is the commonest injury to this coat of the eyeball. It is always the result of external violence, such as a blow, a kick, or a fall, in which the eye is struck with great force, causing sudden change of form. The whole eyeball is driven backwards, but the optic nerve remains stationary; the result is that circular folds are produced in the choroid concentric with the disc, and a rupture takes place at one of these folds. The accident is usually followed by hæmorrhage into the vicinity of the wound, causing opacity of the vitreous, and consequent blindness. This at first prevents the choroidal lesion from being seen with the ophthalmoscope. After a few days, however, as the blood becomes absorbed, vision improves, and a whitish line can be seen in the fundus, immediately opposite to that part of the globe which received the blow. There is, as a rule, a little blood clinging to the edges of the rupture for some time after the latter is visible; but finally the rupture appears as a permanent white or yellowish-white line. It is generally curved in a direction concentric with the edge of the disc, and on its outer side. Occasionally it runs obliquely, and still more rarely it may be equatorial, passing horizontally outwards and inwards on each side of the disc. It frequently bifurcates, and is sometimes multiple. Subsequent to the rupture, there is usually a tendency for masses of pigment to appear round the exposed sclera. Fig. 2, opposite p. 219, shows a painting from a boy who received a blow on his eye from a stick.

Detachment of the Choroid.—See p. 219.

Concussion Choroiditis.—Rupture of the choroid is often accompanied by macular choroidal degeneration. This is of a fine pigmentary nature, and is sometimes termed *Haab's macular disease*.

Rupture of one or more of the choroidal vessels may produce extensive hæmorrhage into the vitreous chamber. (See p. 418.)

NON-PENETRATING INJURIES OF THE RETINA.

Injuries to the retina are almost always the result of concussion of the eyeball, though occasionally exposure to a very bright light has been known to set up a retinitis.

Retinal hæmorrhage is often caused by a blow on the eyeball, and may be small and localised (see p. 418), or may be extensive and cause a large vitreous hæmorrhage (see p. 280).

Retinal detachment is described at length under that heading (see p. 295).

Retinal Rupture is extremely rarely found unaccompanied by choroidal rupture, described on p. 635.

Commotio Retinæ.—As a result of a severe blow on the eye, a somewhat rare condition of the retina is found, with or without other pathological changes. The symptoms complained of are sudden loss of visual acuity, and the gradual production of ‘black specks before the sight.’ If an ophthalmoscopic examination be made immediately after the injury, the fundus reflex is visible, though somewhat diminished in brightness, while the fundus details are seen, as it were, through a thin white cloud. A day or two after the injury this cloud may have become more opaque, the fundus details obscured, and the reflex almost absent. In a few days, however, the opacity begins to be absorbed, and the vision gradually is restored to the normal. *Commotio retinæ* is very commonly accompanied by slight circumcorneal redness, and some spasmodic contraction of the pupil.

The exact pathology of the condition is not definitely known. It is probably due to the entrance of some of the vitreous fluid into the nerve-fibre layer of the retina; some, however, have thought that an œdema of the retina secondary to a subchoroidal hæmorrhage is the cause.

All the treatment that is required is rest to the eyes and shading from bright lights. Atropine and neutral-tinted glasses will secure these.

Retinitis may be produced by a concussion of the eyeball, in which case it is accompanied by choroiditis. (See p. 635.)

‘**Holes**’ at the **Macula**.—This is one of the rarer results

of concussion injuries of the eye. A small circular red depressed area is seen in the macula with clean-cut edges. On the floor of the 'hole' yellowish spots are usually to be seen, and in its immediate neighbourhood the retina is, apparently, in an cedematous and folded condition. A superficial total retinal detachment is sometimes found as an accompanying lesion. Menteith Ogilvie¹ has collected fifteen cases of 'holes' at the macula.

Injuries to the Retina produced by Strong Light.—Ophthalmoscopic changes are the exception rather than the rule in this class of injuries, which are consequently described in the chapter on Amblyopia (p. 337), to which the reader is referred.

NON-PENETRATING INJURIES OF THE LENS.

Concussion cataract is usually, if not always, produced by a rent in the capsule of the lens. This almost always takes place in the immediate neighbourhood of the equator. The consequence of this is that the aqueous has great difficulty in making its way between the lenticular fibres, the edges of the rent come together again, and the cataract usually remains partial. A few cases of rents near the posterior pole have been recorded; here the vitreous takes the place of the aqueous in producing the opacity. It may be only after an interval of several days that this form of cataract shows itself, and consequently the prognosis after a severe blow on the eyeball must be guarded. It is frequently found that other lesions accompany concussion cataract—viz. ruptured choroid, dislocated lens, injury to the iris, and retinal detachment. If the rent in the lens be extensive, secondary glaucoma is liable to supervene.

In most cases the treatment should be conservative. Later, when the eye is quiet, the question of operative interference must be carefully considered, and the answer will depend on the occupation, age, and habits of the patient, the condition of the other eye, and the extent of the cataract. If secondary glaucoma, however, has occurred, immediate operation is imperative. A corneal section must be made, and as

¹ *Trans. Ophth. Soc.* vol. xx. p. 202.

much of the lens substance as possible evacuated ; if necessary, an iridectomy must be performed.

Dislocation of the Lens.—See p. 409.

NON-PENETRATING INJURIES OF THE OPTIC NERVE.

Optic-nerve Atrophy.—Sudden blindness may follow a severe blow on, or in the immediate neighbourhood of, the eyeball, with, for the time being, no ophthalmoscopic signs. After two or three weeks, atrophic changes appear in the disc, and in a short time a picture of total optic atrophy is formed. These cases are usually due to fracture of some part of the bony walls of the orbit, the optic canal being involved. The nerve is compressed or lacerated, with consequent atrophy ; it may even be completely torn across. On account of the interval of time that elapses between the accident and the onset of ophthalmoscopic signs, these cases are important from a medico-legal point of view.

Hæmorrhage into the optic-nerve sheath may be caused by a blow on the head, or by a fall. Several cases have been recorded where sudden blindness came on a day or two after a fall, with, subsequently, gradual return of vision. Other cases of hæmorrhage into the sheath probably end in optic atrophy, and it is difficult to distinguish these from cases of fracture of the orbital bones with damage to the optic nerve.

PENETRATING INJURIES OF THE EYEBALL.

All penetrating injuries of the eyeball, however slight in appearance, must be looked upon as serious, and a guarded prognosis must at first be given. They are recognised by the following points: (1) The tension of the eye is almost always reduced. (2) If the wound is corneal or at the sclero-corneal junction, the anterior chamber is shallow or even obliterated. (3) In many penetrating injuries, the intra-ocular contents are seen projecting between the lips of the wound, iris if the wound is corneal, vitreous if scleral.

The gravity of penetrating injuries depends largely upon

the danger of infection. Apart from this, the severity of the injury is gauged by its extent and position.

Most injuries are punctured or incised wounds, rarely lacerated. They may be divided into two great classes—(a) *Penetrating wounds without the retention of foreign bodies*, and (b) *Penetrating wounds with the retention of a foreign body*.

PENETRATING WOUNDS WITHOUT RETENTION OF FOREIGN BODY.

Cornea.—Penetrating wounds of the cornea are of great importance, on account of the grave complications which sometimes attend them. Their cause is manifold. Children are especially liable to this accident. Attempts are perhaps being made to untie a bootlace with a fork: the fork slips, and is carried violently against the cornea. A nail is being loosened with a screw-driver, which is suddenly jerked upwards and wounds the eye. A fall downstairs while the child is carrying some glass or crockery is another common instance of this accident. As a result, the aqueous humour is tapped, and, with very few exceptions, the iris is at the time of the accident washed out by the aqueous between the lips of the wound. Even if there is no actual prolapse, the iris is approximated to, if not brought into actual contact with, the posterior surface of the cornea.

The corneal wound is frequently complicated by perforation of the iris and lens, and consequently its gravity greatly increased.

In all cases, the cause of injury should be carefully ascertained in order to be sure that no foreign body has entered the eyeball. Should pyogenic organisms enter the eye at the time of the injury, suppurative iritis is very liable to be set up, and panophthalmitis result.

Treatment.—Strict antiseptic measures must be taken, and the eye and patient kept at rest. The patient should be put to bed, and the wound carefully cleansed with perchloride of mercury or chinosol. If the wound shows any signs of being infected, it is often wise immediately to cauterise its edges.

When the iris is entangled or protruding from the wound, the case is more serious. If seen within a few hours after the accident, an attempt should be made to return it. Bearing in mind that the anterior chamber is now quite shallow, we must be careful not to wound the crystalline lens, which is immediately behind the iris. For returning the hernia of the iris, a blunt-ended spatula (fig. 35) may be used, combined with the local use of atropine or eserine, according as the wound is central or peripheral.

It is often found impossible to effect a return of the iris in this manner; in which case the protruding portion should be seized with forceps and snipped off with scissors on a level with the surface, and the edges of the wound freed as much as possible from the iris to prevent the formation of a synechia. Atropine must be instilled into the eye, which should be covered by a light compress, and the patient must be kept in a dark room. These cases often do well, very useful vision being regained.

Other results of penetrating wounds of the cornea are implantation cysts and serous cysts of the iris. (See p. 225.)

Iris.—Penetrating injuries of the iris, without involving the lens, are rare, and need no further description.

Lens.—Penetrating wounds of the lens usually result in total cataract. If the rupture of the anterior lens capsule is slight, the lens fibres swell up, and secondary glaucoma is very liable to arise. If, on the other hand, the rupture be extensive, the lens substance will come forward into the anterior chamber and be slowly absorbed. The treatment consists in aiding the absorption of the lens by needling or extraction of the nucleus, and in performing a paracentesis if glaucoma supervenes. Occasionally the opacity produced is only local.

Sclera.—Rupture of the sclera occasionally results from a severe blow upon the eye. The violence of such an injury is usually sufficient to produce other lesions of the deeper structures. A blow upon the cornea coming directly from the front of the eye generally produces rupture in the region of the equator of the globe. The more common position for the eye to be struck is below the cornea in a direction upwards and

backwards; this, as a rule, produces rupture of the sclera about 2 to 4 mm. above the upper corneal margin and concentric with it.

Such wounds are usually irregular. They are caused by blows with the fist, a stick, stone, racquet-ball, or door-handle. The rupture is generally complicated by injury of the subjacent structures. Extensive wounds are often associated with escape or protrusion of the contents of the globe and total collapse of the eye. Ruptures near the equator are usually attended with more or less hæmorrhage from the choroid into the vitreous chamber; this may be slight, in which case the blood is visible by means of the ophthalmoscope, or it may be so extensive as to prevent all reflection from the fundus, and thus prevent any of its details being seen. When the rupture occurs in the anterior portion of the sclera, the ciliary body is in jeopardy and may be protruding from the wound; the iris may also be drawn outwards with the ciliary body; the crystalline lens may be partly or entirely dislocated, and, indeed, may have escaped from the globe altogether; in some cases the lens becomes lodged outside the globe beneath the ocular conjunctiva. The vitreous humour may be more or less protruding from the wound, and will be recognised by its clear viscid nature. The conjunctiva, owing to its elasticity, may have escaped laceration; this is always a favourable coincidence, inasmuch as the entire membrane protects the lacerated globe from possible septic infection.

Penetrating wounds of the sclera are most common in the anterior portion of the globe between the margin of the cornea and the equator. They are often sclero-corneal, when they usually involve the iris, resulting in prolapse of that structure. If situated within 5 mm. of the sclero-corneal junction—*i.e.* in the so-called 'danger zone'—the ciliary body is almost invariably injured (see below).

Contusion with rupture of the sclera is always of a grave nature. There is not only a lacerated wound of this outer tissue of the globe, but its contents are necessarily much injured and disturbed. Healing of such wounds can hardly be expected without much inflammatory trouble, especially

when the conjunctiva is also involved in the laceration. If, however, there is a possibility of some useful sight being retained, an attempt to save the eye may be made. After antiseptic irrigation, the conjunctiva should be sutured over the wound. In all cases, however, it must be remembered that the retention of the eye is fraught with the danger of inflammation in itself, and of causing sympathetic ophthalmitis in the other eye.

PENETRATING WOUNDS WITH RETENTION OF FOREIGN BODY.

The entrance into the eyeball of a foreign body is an extremely serious accident, and only too often leads to the destruction of the eye. The eye is, as it were, surrounded by many pitfalls, into any one of which it may be hurled sooner or later. The diagnosis is by no means easy, the localisation of the foreign body often impossible, the damage done to the tissues of the globe often irreparable, the foreign body may be accompanied by pyogenic organisms, or it may give rise to chemical changes the products of which set up acute inflammation; and, lastly, the other eye may be in constant danger of sympathetic inflammation.

The commoner foreign bodies found in the eyeball are splinters of iron, fragments of copper, lead shot, flint, glass, and wood. Hairs have been carried into the eyeball, either as a result of operation, or of a perforating wound of the cornea. Workers in the iron and steel trade are very liable to get iron and steel splinters in their eyes, especially during the processes termed 'chipping' and 'fettling.' Again, the explosion of a percussion cap, or the ricochet of a shot, frequently results in the lodgment of a piece of copper or a lead pellet within the cavity of the globe. Less commonly a fragment of glass may enter the eye, the accident being the bursting of a soda-water bottle.

Symptoms.—The signs of penetration of the globe are necessarily present (see p. 638). The wound of entrance of the foreign body either in the sclera or cornea is, as a rule, visible, though occasionally, when the case is not seen until

some time after the accident, a small corneal nebula or scleral scar is with difficulty discovered. In almost all cases there is more or less pain; signs of inflammation in the eye generally appear and increase in intensity. The continued presence of a foreign body almost invariably leads to the destruction of the eye, though this may not take place for months, or even years, after the accident. It is true that a small, thoroughly aseptic and non-irritating particle may enter the eye and remain there for a long period without causing more than a slight disturbance of the visual field or visual acuteness. In the majority of cases, however, inflammation follows immediately after the accident. The inflammation takes the form either of plastic irido-cyclitis, or of panophthalmitis, and is particularly liable to set up sympathetic inflammation.

The Diagnosis and Localisation of the Foreign Body.—The diagnosis of the presence of a foreign body within the globe is of the greatest importance, and in all penetrating wounds of the sclera and cornea must be carefully considered.

By the *history* of the case it is often possible to decide whether a foreign body is within the eye or not. Sudden pain in the eye, as if struck with something, while out with a shooting party, followed by some hæmorrhage, combined with the signs of perforation, make the diagnosis of a spent shot within the globe almost certain.

It is unusual for the foreign body to be *visible to the naked eye*. Should it, however, lodge in the iris, it may be seen lying on its surface. It may become embedded within the substance of the crystalline lens, and so be visible by oblique focal illumination.

The *ophthalmoscope* may discover a foreign body. If the case is seen very soon after the injury, the piece of steel may be found in the vitreous or on the retina. This, however, is rarely the case, as haziness of the surrounding media due to hæmorrhage, inflammatory changes, or opaque lens, obscures all details. Occasionally, months or years after an accident, the foreign body has been seen with an ophthalmoscope floating in the vitreous or lying upon or embedded in the superficial layers of the retina of an otherwise normal eye.

Should an opaque stria be seen in the vitreous combined with a corneal nebula, an opacity in the lens substance or capsule, and a history of injury, a foreign body must be suspected.

The *electro-magnet* is of great use in the diagnosis, as well as in the treatment, of magnetic foreign bodies in the eyeball. The practical utility of this instrument has been amply proved in the practice of Snell, Hirschberg, McHardy, Bradford, Haab, and others. The instrument (fig. 189) consists of a

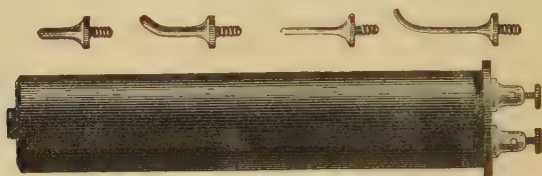


FIG. 189.—Snell's Electro-magnet.

core of soft iron, around which is placed a coil of insulated copper wire, the whole being enclosed in an ebonite case. At one end are two screws to receive the battery connections; at the other end the core of the magnet projects in such a manner that one of the nozzles represented in the figure can be screwed into it. Two methods may be used. The nozzle of a small electro-magnet may be introduced through the original wound, and the foreign body may be extracted or moved; in the latter case it will sometimes give a distinct sense of discomfort or even pain. More certain, however, is the giant magnet of Haab.¹ No nozzle is introduced into the wound, but the eye is brought into the immediate neighbourhood of one of the conical points of the instrument. In this way foreign bodies may be removed from the vitreous and brought into the anterior chamber, or even out through the original wound. In other cases, acute pain is felt by the movement of the embedded particle.

The *sideroscope* is a magnetic needle, combined with a telescope for observing any deflection. A magnetic body in

¹ Haab's Giant Magnet, with switch, rheostat, and stout frame, may be obtained (price 30*l.*) of K. Schall, 35 Great Marylebone Street, London, W. It is wound either for 100 or 200 volt supplies.

the eye will cause a deviation of the needle if the sideroscope is placed near the eye.

But of all the means that can be used for the diagnosis and localisation of foreign bodies within the eyeball, *skiagraphy*, by means of the Röntgen rays, is the most important. This method is closely associated with the name of Mackenzie Davidson. The principle on which he works is to take two stereoscopic photographs, and so get the position of the foreign body in space; the co-ordinates of this position are measured and compared with those of a known fixed point, and thus the exact position and size of the foreign body can be determined to within one-fiftieth of an inch. The apparatus consists of two essential parts, the photographic box and Crookes tube, and the localiser. The patient's head is firmly fixed laterally against two taut piano-wires at right angles to each other in the vertical plane. The Crookes tube, which should have an anode of osmium, and be placed at a distance of 30 cm. from the wires, is in such a position that the anode is exactly opposite their intersection, as determined by a rifle sight. Just below the eye to be examined (which is always placed towards the wires) is fastened a piece of lead, 1 cm. long, the position of which relatively to the cornea must be accurately measured. The optic axis of the eye must be kept parallel to the horizontal wire. A sensitive plate is placed vertically against the wires. The Crookes tube, which can be moved in either direction horizontally, is displaced through 3 cm., and a photograph is taken. The plate is replaced by another, the tube displaced through 3 cm. in the opposite direction, and a second photograph is taken. An exposure of from ten to sixty seconds is required. The developed negatives may be examined in a stereoscope, whereby a fairly accurate estimate of the position of the foreign body may be determined. To exactly measure its size and position a localiser is necessary. A tracing of the positions of the lead and foreign body in the two negatives is taken on a thin piece of celluloid with two cross lines at right angles upon it, the cross lines of the piano-wires on the negatives coinciding with these lines on the celluloid. This tracing is placed on the localiser, which consists of a horizontal glass plate and two threads,

representing exactly the paths of the X-rays in the two positions of the Crookes tube. Their intersection marks the position of the foreign body in space, and all that is necessary is to measure the co-ordinates of this position, and those of the fixed known point, namely the upper end of the lead. Not only are all metals opaque to the X-rays, but most varieties of glass throw a shadow, though less distinct.

Prognosis.—Owing to the recent additions to the means of diagnosis and treatment, the prognosis is not so bad as formerly. It is, however, usually impossible to exclude the possibility of sepsis, and it is largely for this reason that, even though the foreign body be successfully and quickly removed, a guarded prognosis must always be given. Within the last few years many eyes have been saved, and some useful vision retained. The earlier the foreign body is removed, the more hopeful the prognosis. If the foreign body remains in the eyeball, we find one of the following conditions: (1) The eye may, though very rarely, tolerate the foreign body, and all signs of inflammation disappear, with restoration of much useful sight; (2) localised inflammation, of a more or less chronic form, may set in, followed by contraction of the vitreous and detachment of the retina, with final atrophy of the whole globe; (3) acute inflammation may occur, resulting in panophthalmitis; (4) the foreign body may remain for some time visible and movable in the vitreous cavity, and whilst in this condition it may bring on a glaucomatous attack in this eye, or produce sympathetic inflammation in that of the opposite side.

Treatment.—*Prophylactic.*—Owing to the extreme gravity of the presence of a foreign body in the eyeball, the prophylactic treatment is of great importance. As mentioned above, it is amongst workers in iron and steel that this accident is most prone to occur, and it is in these trades that much can be done to guard against this form of injury. The flying about of the iron splinters is a constant source of danger, and, consequently, as much space as possible should be allowed between the men. The pneumatic chipper scatters the fragments much less than other forms. But most important of all is the use of protectors. Those engaged in

grinding should wear glass protectors with gauze sides, while the chippers must substitute for glass, fine-meshed aluminium or galvanised-iron protectors.

Operative.—Having diagnosed the presence of a foreign body, and having localised it as far as possible, it must, with the few exceptions mentioned below, be immediately removed.

If it is free in the anterior chamber, and magnetic in properties, it may be removed by the electro-magnet, the nozzle of which is placed into the original wound. Or a corneal incision may be made, and it may be extracted with forceps.

A foreign body lodged in the iris is best removed through an incision at or near the periphery of the cornea. If magnetic, the nozzle of an electro-magnet should be introduced through the incision. If non-magnetic, or if the electro-magnet fail, that portion of the iris in which the particle is situated should be seized with forceps, drawn through the corneal incision, and an iridectomy performed in such a way that the foreign body is included in the excised portion of the iris. It is extremely difficult to pick a foreign body off the iris without causing hæmorrhage, which renders it invisible, and thus complicates the treatment.

Foreign bodies in the deeper parts of the eyeball, viz. ciliary body, vitreous humour, retina, choroid, and sclera, demand the utmost care and precision in their removal. The patient should, if possible, be brought into the immediate neighbourhood of Haab's giant magnet, when the particle, if magnetic, may be brought into the anterior chamber and then removed by incision. If the magnet fails, and the particle is invisible, two skiagrams should be taken (as described above), and the exact position of the foreign body determined. Under complete general anæsthesia the wound should be enlarged with a Graefe knife, and the electro-magnet, or the forceps, directed towards the known site. In the former case, a click will usually be heard as the body and the magnet come in contact. Great care must be exercised during the withdrawal. Finally, antiseptic dressings are applied. There are two modifications of the operation. A fresh incision in the horizontal direction and between two recti muscles may be made either in the immediate neighbourhood of the foreign body,

or opposite to its position. In these cases, after removal, the wound should be closed by a conjunctival suture.

When the foreign body is non-magnetic, such as pellets of lead or copper, the only chance of saving the eye is to cut down upon the foreign body and remove it with forceps; but success is rare.

There are certain contra-indications against immediate removal.

The foreign body may become embedded within the substance of the crystalline lens. One of two things may happen. The lens may become rapidly opaque and swollen, causing an acute secondary glaucoma, in which case it must be removed at once. Or the eye may quieten down and the cataractous condition of the lens slowly progress, in which case removal of the foreign body should be postponed until the cataract is complete. The lens and foreign body are then extracted together.

Occasionally patients are seen some weeks or months after the accident, with the eye quiet though possessing within it a foreign body. The inflammatory exudate which was formed around it at the time has become organised into a cystlike casing; when thus encysted it may be tolerated for an indefinite time without pain, and the vision may be to a great extent restored. Metallic substances are extremely unlikely to be thus tolerated, even though they enter the eye in an aseptic condition, since the products of chemical action upon them cause irritation and even inflammation; this is especially the case with regard to copper. These cases of quiet useful eyes containing a foreign body with no sign of active inflammation should not be touched; they must, however, be carefully watched, as inflammatory signs may set in months or even years after the accident.

The third contra-indication against attempted removal of the foreign body is complete disorganisation of the eyeball by the accident. In such a case enucleation should at once be performed.

In all cases of foreign body within the globe, strict watch must be kept upon the other eye for any sign of threatening sympathetic ophthalmitis.

INJURIES OF THE ORBIT.

Injuries of the Orbital Walls.—Fracture of one or more of the walls of the orbit may arise from a severe blow on the eyeball, from a penetrating injury, or from a gunshot wound. Its results may be slight or severe. Communication is usually set up with one or more of the air spaces in the immediate neighbourhood, namely, the frontal and ethmoid sinuses, the antrum of Highmore, and the nasal passages. Emphysema of the lids or of the cellular tissues of the orbit is a pathognomonic sign of this accident (see p. 611). Fragments of bone from a comminuted fracture, especially of the roof of the orbit, are liable to compress or lacerate the optic nerve, causing sudden blindness; ophthalmoscopic changes appear only some few weeks later.

Injuries of the Orbital Nerves and Muscles.—The orbital muscles may be injured directly by penetration of the orbit by a foreign body, the injury even amounting to complete division of all the muscle fibres. Similarly, though of extreme rarity, one of the orbital nerves, usually the branch to the external rectus, may be ruptured. As a result of either of these injuries, a strabismus immediately following an injury is produced. The accident is often complicated by intra-orbital hæmorrhage, and the optic nerve itself may be damaged or ruptured. Occasionally it is possible to reunite a divided muscle produced in this way.

More frequently, however, this class of orbital injuries is produced indirectly. An intra-orbital hæmorrhage is very liable to subsequently produce adhesions, which involve muscles and nerves, and so cause impairment of mobility of the eyeball. Again, in the repair of a fracture of part of the orbital walls, the resulting callus may press upon one or more of the nerves supplying the orbital muscles, and so produce diplopia and strabismus. In these cases little can be done to hasten the slow subsidence of the symptoms which usually takes place.

Intra-orbital Hæmorrhage. — Most injuries to the orbit result in more or less intra-orbital hæmorrhage, producing the symptoms of this condition (see p. 610). Fracture of

one of the orbital walls is the commonest cause, though simple rupture of a blood-vessel in the post-ocular tissues may be the only lesion resulting from a blow on, or in the neighbourhood of, the eyeball.

Foreign Bodies within the Orbit.—The orbit is not a very uncommon situation for a foreign body to become embedded in, while owing to the amount of fat which the orbit contains, its presence may be unsuspected for many days—hence the importance of making a very careful examination of a wound in the eyelid or conjunctiva. As an instance of the ease with which a large foreign body may be concealed in the orbit, the case published by Carter is probably unique. An old man fell, while drunk, down a flight of steps, at the bottom of which was a row of hat-pegs. He received a contusion, and a cut on the eyelid, which, after a few days, induced him to seek advice; a surgeon treated him for several days, and then noticed a black substance lying in the wound; on seizing this with forceps he succeeded in withdrawing the shaft of a hat-peg measuring $3\frac{1}{4}$ inches in length.¹

The foreign body may pass between the globe and the orbital wall, lodging in the orbital fat. It may, however, pass right through the globe, tear or sever the optic nerve, fracture part of the bony wall, and even enter the brain. It may cause infection of the injured parts, setting up meningitis, and ending in the death of the patient.

The passage of a bullet through the orbit often gives rise to extensive intra-ocular changes, although the globe has escaped any direct injury. The rapid transit of the bullet sets up powerful vibrations, which act as 'secondary missiles' (W. F. Stevenson). These indirect lesions are vitreous and retinal hæmorrhages, retinal detachment, choroidal rupture, and adhesive choroido-retinitis. In addition, the intra-ocular tension may be lowered, although there is no perforation of the globe.

Displacements of the Globe.—*Exophthalmos* as a result of injury may be produced in various ways. A severe hæmorrhage into the orbital tissues causes marked proptosis, as also does an infective cellulitis with considerable exudation. A

¹ The peg is in St. George's Hospital Museum.

perforating injury may sever many of the orbital muscles, with protrusion of the globe, perhaps even in front of the eyelids. Lastly, the form known as pulsating exophthalmos (see p. 621) has as its commonest cause a severe injury to the head.

Enophthalmos due to injury is very rare, and its pathology is somewhat obscure. It may follow immediately, or some time after, a severe blow on the eyeball, such, for instance, as would be produced by the shaft of a cart. It is probably the result of a fracture of the floor of the orbit, and in some cases the eyeball partially enters the antrum of Highmore. Other causes that may give rise to traumatic enophthalmos are atrophy of the orbital fat, and retraction of orbital fibrous tissue the product of inflammation. In slight cases the vision is usually unimpaired, if no other lesion be present. To detect slight cases the observer should stand behind the patient, who is sitting, and raise the upper lids, directing the patient at the same time to look down; the relative prominence of the two eyes can in this way be accurately determined.

Luxation of the eyeball is of extreme rarity. The globe may be luxated forwards in front of the eyelids, or backwards and downwards into the antrum of Highmore. As a rule, the optic nerve is either lacerated or completely torn through. Enucleation is generally necessary.



APPENDICES.

APPENDIX I.

FORMULÆ.

CAUSTICS.

No.

1. Argenti nitratis gr. v, vel gr. x, vel gr. xx; aq. destill. ℥j.
Dissolve.

To be brushed on the everted lids in granular or catarrhal conjunctivitis, the excess being washed away with salt solution.

2. Hydrargyri perchloridi gr. j (dissolve in glycerine, q. s.); aq. destill. ℥j (1 in 500).

To be brushed on the everted lids in granular conjunctivitis. Cocaine should be used before and after, as the application is otherwise very painful.

3. Lapis divinus. Made by fusing together equal parts of sulphate of copper, alum, and nitrate of potash, with camphor $\frac{1}{10}$ part of the whole added, and running into moulds.

Used in chronic granular or catarrhal conjunctivitis.

4. Mitigated nitrate of silver crayon is made by fusing together equal parts of nitrate of silver and nitrate of potash, and running into moulds.

5. The same, consisting of 1 part nitrate of silver, and 2 parts nitrate of potash.

6. The same, consisting of 1 part nitrate of silver, and 3 parts nitrate of potash.

7. The same, consisting of 1 part nitrate of silver, and 4 parts nitrate of potash.

8. Protargol solution 2 per cent. to 30 per cent.

Used in all forms of conjunctivitis, and in lachrymal obstruction.

DROPS.

No.

- 8A. Adrenalin chloride solution (Parke, Davis, & Co.), 1 in 2000.

A powerful hæmostatic. Used for blanching the conjunctiva in conjunctivitis, &c.; also used to render operations in ophthalmic surgery almost bloodless. It increases the anæsthetic action of cocaine.

9. Argenti nitratis gr. ss to gr. j; aq. destill. ℥j. Dissolve.
 10. Atropinæ sulphatis gr. ij to gr. iv; aq. destill. ℥j. Dissolve.
 11. Atropinæ sulphatis gr. iv; cocainæ hydrochloratis gr. xx; aq. destill. ℥j. Dissolve.

Used where a very powerful mydriatic is desired: *e.g.* to break down posterior synechiæ in iritis.

12. Cocainæ hydrochloratis gr. v to gr. xx; aq. destill. ℥j. Dissolve.

Mydriatic and local anæsthetic; used in most operations, and in painful corneal wounds. It may be simply instilled into the palpebral sac, or injected beneath the conjunctiva of the globe or beneath that of the eyelid by means of a hypodermic syringe.

13. Cupri sulphatis gr. ss to gr. ij; aq. destill. ℥j. Dissolve.

14. Daturinæ sulphatis gr. ij; aq. destill. ℥j. Dissolve.

15. Duboisinæ sulphatis gr. j; aq. destill. ℥j. Dissolve.

16. Eserinæ sulphatis gr. j to gr. ij; aq. destill. ℥j. Dissolve.

Miotic. Used in primary glaucoma, and in peripheral wounds of the cornea.

17. Eserinæ sulphatis gr. ij; cocainæ hydrochloratis gr. xx; aq. destill. ℥j. Dissolve.

Used where anæsthesia is desired with a contracted pupil: for example, in operations for cataract.

18. Fluorescini gr. viij; liquoris potassæ m 180; aq. destill. ℥j. Dissolve.

Used as an aid to the diagnosis of an abrasion of the cornea, or a small corneal ulcer.

19. Homatropinæ hydrobromatis gr. iv to gr. vj; aq. destill. ℥j. Dissolve.

Used where the mydriatic effect is required only for a short time, as in the ophthalmoscopic examination of the fundus, vitreous, or lens. Also used in estimating errors of refraction in adults.

20. Homatropinæ hydrobromatis gr. iv; cocainæ hydrochloratis gr. x; aq. destill. ℥j. Dissolve.

Action similar to that of homatropine alone, but quicker and more powerful.

No.

21. Hyoscinæ hydrobromatis gr. j to gr. ij ; aq. destill. ℥j. Dissolve.

This, and Nos. 14 and 15, are similar in action to, and intermediate in strength between, homatropine and atropine, and are useful in the same cases. They may be tried as substitutes for atropine where that drug causes irritation.

22. Pilocarpinæ nitratis gr. ij ; aq. destill. ℥j. Dissolve.

Miotic. Used in the same class of cases as eserine ; it is less powerful and less irritating.

23. Zinci chloridi gr. ss to gr. ij ; aq. destill. ℥j. Dissolve.

Used in many forms of chronic conjunctivitis, where more energetic measures are indicated than the lotions 28, 29, and 35.
(To be avoided if there is any affection of the cornea.)

FOMENTATIONS.

24. Extracti belladonnæ ʒj ; aquæ Oj. Dissolve.

To be used as a fomentation in acute iritis, cyclitis, &c.

25. Acidi borici ʒvj ; aquæ Oj. Dissolve.

To be used as a fomentation in orbital cellulitis, styes, and other affections where moist heat is desired.

HYPODERMIC INJECTION.

26. Pilocarpinæ nitratis gr. iij ; aq. destill. ʒj. Dissolve. Two or three minims to be injected hypodermically daily, the dose being gradually increased up to six minims.

The object is to produce profuse perspiration and slight salivation. Used in detached retina, choroiditis, retinitis, and iritis.

INFUSION.

27. Infusum abri. Take of powdered jequirity seeds ʒj ; water at 120° F., ʒxij ss. Let stand till cool, and decant.

Used in granular ophthalmia with total pannus. (See p. 119.)

LOTIONS.

28. Acidi borici gr. x ; aq. destill. ℥j. Dissolve.

29. Aluminis gr. ij to gr. v ; aq. destill. ℥j. Dissolve.

30. Chinosol gr. $\frac{1}{4}$; aq. destill. ℥j (1 in 2000). Dissolve.

Used in acute catarrhal and purulent conjunctivitis ; also before and after operations.

No.

31. Hydrargyri perchloridi gr. $\frac{1}{10}$; aq. destill. $\mathfrak{z}\text{j}$ (1 in 5000).
Dissolve.

Used in purulent ophthalmia; sometimes in granular and catarrhal conjunctivitis; also before and after operations.

32. Plumbi subacetatis gr. ij; aq. destill. $\mathfrak{z}\text{j}$. Dissolve.

Used in chronic conjunctivitis.

33. Sodii biboratis gr. x; aq. destill. $\mathfrak{z}\text{j}$. Dissolve.

Used in blepharitis, to wash away the crusts.

34. Sodii bicarbonatis gr. x; aq. destill. $\mathfrak{z}\text{j}$. Dissolve.

Used in burns by acids; also in blepharitis.

35. Zinci sulphatis gr. ss to gr. ij; aq. destill. $\mathfrak{z}\text{j}$. Dissolve.

Used in chronic forms of conjunctivitis.

OINTMENTS.

36. Acidi borici gr. x; vaselini albi $\mathfrak{z}\text{j}$. Mix.

Used in conjunctivitis and blepharitis.

37. Atropinæ (alkaloid) gr. ij to gr. viij; vaselini albi $\mathfrak{z}\text{j}$. Dissolve by warmth.

38. Atropinæ gr. ij to gr. iv; cocainæ gr. x; vaselini albi $\mathfrak{z}\text{j}$.
Dissolve by warmth.

This and No. 37 used similarly to the drops 10 and 11. It is often easier for parents, &c., to put ointment into children's eyes than drops.

39. Hydrargyri oxidi flavi gr. viij or less; vaselini albi $\mathfrak{z}\text{j}$. Mix.

Used in phlyctenular conjunctivitis; also in corneal nebulæ as a massage.

40. Hydrargyri ammoniati gr. xxv; vaselini albi $\mathfrak{z}\text{j}$. Mix.

Used in blepharitis.

41. Ung. hydrargyri nitratis gr. xl; vaselini albi $\mathfrak{z}\text{j}$. Mix.

Used in blepharitis, in the same cases as No. 42.

42. Ung. hydrargyri oxidi rubri $\mathfrak{z}\text{j}$; vaselini albi $\mathfrak{z}\text{j}$. Mix.

Used in blepharitis and conjunctivitis; also in corneal nebulæ as a massage.

43. 'Ung. Simplex.' Ceri albi $\mathfrak{z}\text{j}$; adipis præparati $\mathfrak{z}\text{ij}$. Mix.

Rubbed along the edges of the lids at night in conjunctivitis, to prevent the lids sticking together.

APPENDIX II.

TESTING VISION FOR THE SERVICES.

1. *The Army.* (a) *Commissioned Officers, including the Royal Army Medical Corps.*—The minimum standard for each candidate is $\frac{6}{24}$ Snellen with each eye without glasses, $\frac{6}{6}$ in one eye and $\frac{6}{12}$ in the other when properly corrected with suitable glasses; at the same time, he must be able to read D=0·8 with each eye without glasses, at any distance selected by himself. Strabismus, inability to distinguish the principal colours, or any disease in either eye subject to the risk of aggravation or recurrence, disqualifies.

The following riders¹ were issued with the Army Orders dated July 1, 1901 :

‘Normal vision of one eye may be sufficient to allow a higher defect in the other, to the extent of one-sixth, if the defect is simple error in refraction remedied by glasses.

‘The candidate must be able to read the tests without hesitation in ordinary daylight.’

(b) *Recruits.*—Recruits have to recognise the number and position of ‘dots’ on a card at a given distance.

The directions for using the ‘Test Dot Card’ are as follows :

(1) Place the recruit with his back to the light, and hold the test card perfectly upright in front of him, letting the light fall fully on the card.

(2) Measure off with precision 10 feet in the case of a recruit for the Regular Army (including the Army Service Corps), the Militia, Imperial Yeomanry, and Volunteers, and 5 feet in the case of a recruit for the Royal Army Medical Corps, or a Departmental Corps.

(3) Examine each eye separately. The eye not under trial

¹ There are no similar riders in the *Regulations for Admission to the Royal Army Medical Corps.*

should be shaded by the hand of an assistant, who will take care not to press on the eyeball.

(4) Expose some of the 'dots'—not more than seven or eight at a time—and desire the recruit to name their number and positions; vary the groups frequently to provide against deception.

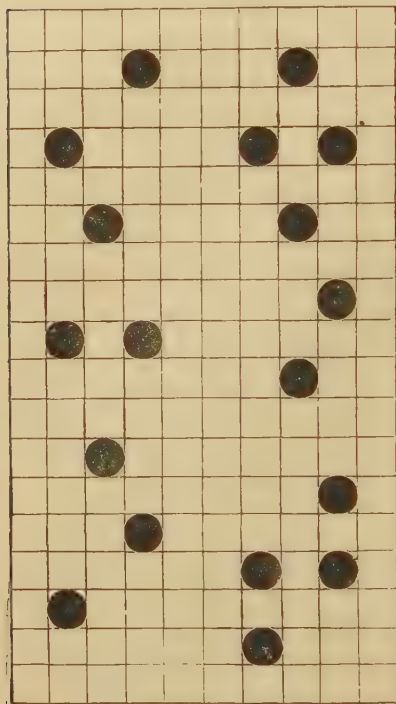


FIG. 190.—The 'Dot' Test.

(5) Recruits for Volunteers may wear spectacles while being tested.

Each 'dot' corresponds, at a distance of 10 feet, with a bull's-eye 3 feet in diameter, at 600 yards.

2. *The Royal Navy.*—Candidates for Naval Cadetships and Engineer Studentships must have full normal vision, both distant and near, in each eye, as determined by Snellen's Test Types. Any defect in colour-sense will disqualify the candidate.

It is not essential that candidates for Naval Surgeonships should possess the full degree of vision, but no standards are specified.

3. *The Home Civil Service.*—Blindness or defective vision disqualifies, except a moderate degree of ordinary

short-sight, but there is no specified standard. If one eye has been lost by mechanical injury, and the other eye is healthy, sufficient, and not likely to become affected, the case will be specially considered after the candidate has passed his examination. Colour-blindness is not in itself a disqualification for ordinary Home appointments, but it is for certain special appointments.

Customs.—Candidates for the outdoor service must not be short-sighted, and Boatmen must not be colour-blind.

Prisons (English).—Good eyesight is necessary, though no standard is specified.

4. *The Indian Civil Service (Covenanted and Uncovenanted).*—If, with proper correction, vision is not less than $\frac{6}{9}$ in one eye, and

$\frac{6}{6}$ in the other, a candidate will be admitted. Morbid changes in the fundus of either eye will disqualify, with the exception of a posterior staphyloma, provided the ametropia of either eye does not exceed 2.5 D, and no signs of activity are present. If a corneal nebula renders the sight of either eye less than $\frac{6}{12}$, the candidate will be disqualified; if the sight of one eye is reduced to $\frac{6}{12}$ by a nebula, the vision of the other eye must equal $\frac{6}{6}$, with or without glasses.

Paralysis of an external muscle disqualifies. Absence of binocular vision is not in itself a disqualification, neither is colour-blindness.

5.. *The Departments (Indian) of Forest, Survey, Telegraph, Factories, and for various Artificers.*—Not more than 2.5 D of myopia is allowed. Myopic astigmatism is not a disqualification, provided the combined sphero-cylindrical correcting glass does not exceed 2.5 D. Not more than 4 D of total hypermetropia is allowed. Hypermetropic astigmatism is not a disqualification, provided the combined sphero-cylindrical correcting glass does not exceed +4 D. In all these cases, the vision, with or without the correcting glasses, in one eye must equal $\frac{6}{9}$ and in the other $\frac{6}{6}$, the range of accommodation being normal. There must be no evidence of progressive fundus disease. If a corneal nebula diminishes the sight of one eye to $\frac{6}{12}$, the better eye must be emmetropic; the candidate is disqualified if the sight of one eye is less than $\frac{6}{12}$, owing to the presence of a corneal nebula.

A candidate must be able to distinguish the principal colours. There must be no paralysis of any one or more of the external ocular muscles.

6. *The Department (Indian) of Public Works.*—The standards closely resemble those of the Forest, Survey, &c. Departments, the only difference being that 3.5 D of myopia is allowed, and the combined sphero-cylinder which corrects any myopic astigmatism may reach 3.5 D.

7. *The Indian Medical Service and the Police Department.*—A candidate must read D=24 at 6 metres and D=0.8 at any distance selected by himself, with each eye without glasses. He must, with the proper correction, read D=6 at 6 metres with one eye, and D=12 at the same distance with the other eye.

Squint, inability to distinguish the principal colours, and any

morbid condition which may either become aggravated or recur, must be absent.

8. *The Indian Pilot Service, and Candidates as Guards, Engine-drivers, Signalmen, and Pointsmen on Railways.*—Both eyes must be emmetropic; vision, range of accommodation, and colour-sense must be perfect. Squint, or any defective action of the extra-ocular muscles, must be absent.

9. *The Indian Marine Service, including Engineers and Firemen.*—Any existing error of refraction must be neutralised by a concave or convex lens, not exceeding 1 D. A candidate must be able to distinguish the principal colours and their various shades. Squint, or any defective action of the extra-ocular muscles, must be absent.

10. *The Royal Irish Constabulary.*—Candidates for cadetships "must be able to read with each eye separately, and without glasses, Snellen's Metrical Test Types (Edition 1898), numbered D=10, at 20 English feet, and those numbered D=0·8 at any distance selected by the candidate himself. Squint, inability to distinguish the principal colours, or any morbid condition liable to the risk of aggravation or recurrence in either eye, will involve the rejection of the Candidate." The same regulations apply to Constables.

11. *The British Mercantile Marine.*—All persons intending to serve in the Mercantile Marine must pass the sight tests before they can be examined for any certificate. The examination consists of three parts: (a) Form Vision Test; (b) Colour Vision Test; (c) Colour Ignorance Test.

(a) *Form Vision Test.*—The candidate is placed at a distance of 16 feet from Snellen's old types, and is expected to read three of the five letters in the fifth row from the top. This corresponds to about $\frac{6}{18}$ of the newer types. He may use both eyes, or either eye, but no spectacles. If he cannot read, he must be tested with sheets of dots placed exactly opposite to him at a distance of 8 feet. Every candidate who fails to pass the Form Vision Test is examined with the Pellet Test. The pellets are of different colours and shades, and this test is performed in the same way as is the Holmgren's Wools Test. Should the candidate pass the Pellet Test, the principal examiner is communicated with for his instructions as to whether the candidate is or is not to be regarded as having failed in Form Vision.

(b) *Colour Vision Test.*—The colour vision of candidates, as tested by Holmgren's wools, must be perfect. A full description of the method used is to be found in the 'Regulations relating to

the Examination of Masters and Mates in the Mercantile Marine,' issued by the Board of Trade, and published by Messrs. Eyre & Spottiswoode, 1900.

(c) Colour Ignorance Test.—This test is confined to naming the three colours, red, green, and white. Pure red and green skeins of wool are used, and any white object, such as white paper. If the candidate makes any mistake in naming the colours, he is tried with a lantern and coloured glasses.

Candidates who fail in the Form Vision Test or the Colour Ignorance Test may be re-examined after an interval of three months; candidates who fail in the Colour Vision Test may not be re-examined.

APPENDIX III.

TEST TYPES.

SNELLEN'S READING-TYPE.

D=0,3 metre.

The Gallic tribes fell off and sued for peace. Even the Batavians became weary of the hopeless contest, while fortune, after much capricious hovering, settled at last upon the Roman side. Had Civilis been successful, he would have been deified; but his misfortunes at last made him odious in spite of his heroism. But

the Batavian was not a man to be crushed. Nor had he lived so long in the Roman service to be out-matched in politics by the barbarous Germans. He was not to be sacrificed as a peace-offering to revengeful Rome. Watching from beyond the Rhine the progress of defection and the decay of national

D=0,6 metre.

enthusiasm, he determined to be beforehand with those who were now his enemies. He accepted the offer of negotiation from Cerialis. The Roman general was eager to grant a full pardon, and to re-enlist so brave a soldier in the service of the empire. A colloquy was agreed upon. The bridge across the Nabalia was broken asunder in the middle, and Cerialis and Civilis met upon the severed sides. The placid stream by which Roman enterprise had connected the waters of the Rhine with the lake of Flevo, flowed between the imperial

D=0,8 metre.

commander and the rebel chieftain. — Here the story abruptly terminates. The remainder of the Roman's narrative is lost, and upon that broken bridge the form of the Batavian hero disappears for ever. His name fades from history: not a syllable is known of his subsequent career; everything is buried in the profound oblivion which now steals over the scene where he was the most imposing actor. The contest of Civilis with Rome contains a

D=1 metre.

remarkable foreshadowing of the future conflict with Spain, through which the Batavian republic, fifteen centuries later, was to be founded. The characters, the events, the amphibious battles, desperate sieges, slippery alliances, the traits of generosity, audacity, and cruelty, the generous confidence, the broken faith, seem so closely to repeat themselves, that history appears to present the

D=1,25 metres.

selfsame drama played over and over again, with but a change of actors and of costume. There is more than a fanciful resemblance between Civilis and William the Silent, two heroes of ancient German stock, who had learned the arts of war and peace in the service of a foreign and haughty world-empire. Determination.

D=1,5 metres.

concentration of purpose, constancy in calamity, elasticity almost preternatural, self-denial, consummate craft in political combinations, personal fortitude, and passionate patriotism, were the heroic elements in both. The ambition of each was subordinate to the

D=1,75 metres.

cause which he served. Both refused the crown, although each, perhaps, contemplated, in the sequel, a Batavian realm of which he would have been the inevitable chief. Both offered the throne to a Gallic prince,

D=2,25 metres.

for **Classicus** was but the prototype of **Anjou**, as **Brinno** of **Brederode**, and neither was destined, in this world, to see his sacrifices crowned with success.

D=3 metres.

The characteristics of the two great races of the land portrayed themselves in the Roman and the Spanish struggle with much

D=4 metres.

the same colors: twice a Batavian republic took its rank among the leading powers of the earth, the

MOTLEY.

APPENDIX IV.

PLATES ILLUSTRATING THE NORMAL AND
PATHOLOGICAL HISTOLOGY OF THE EYE.

(SEMIDIAGRAMMATIC.)

BY GEORGE S. KEELING, M.B., B.C.

PLATE A.

FIG.

1. *Acute Catarrhal Conjunctivitis*, showing epithelial and sub-epithelial infiltration. (See p. 65.)
2. *Follicular Conjunctivitis*, showing a lymphoid follicle and lymphoid infiltration of the subepithelial tissue. (See p. 69.)
3. *Granular Conjunctivitis (Trachoma)*, showing a lymphoid follicle with an incomplete connective tissue capsule. There are no certain histological differences between follicular and granular conjunctivitis. (See p. 84.)

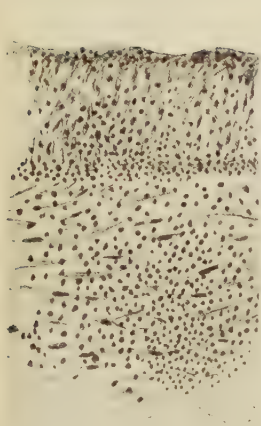


FIG. 1.



FIG. 2.



FIG. 3.

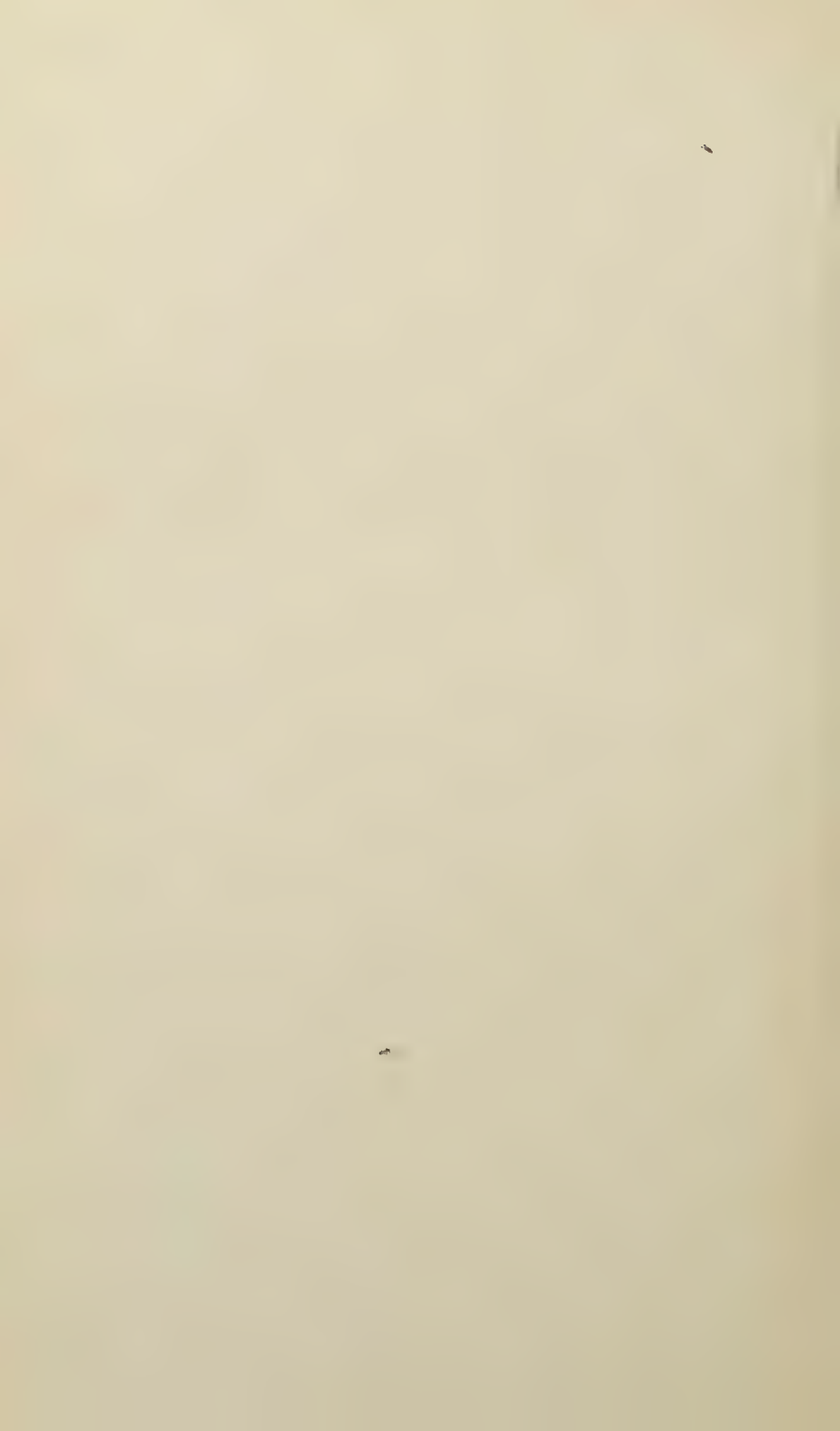


PLATE B.

PLATE B.

FIG.

1. *Normal Cornea*, showing
 - a. Corneal epithelium.
 - b. Bowman's membrane.
 - c. Substantia propria.
 - d. Descemet's membrane and endothelium.
 (See p. 107.)
2. *Vascular Keratitis (Pannus)*, showing a layer of granulation tissue lifting the corneal epithelium from Bowman's membrane. (See p. 117.)
3. *Corneal Cicatrix*, showing cicatricial tissue throughout the whole thickness of the cornea, with dipping down and thickening of the epithelium. Bowman's membrane and Descemet's membrane are both absent from the cicatrix. (See p. 129.)
4. *Interstitial Keratitis*, showing a vascularisation and infiltration of the substantia propria. (See p. 114.)
5. *Keratitis Punctata (Precipitates)*, showing, on the posterior surface of the cornea, a deposit consisting of round cells, pigment granules, and fibrin. The endothelium is absent, though Descemet's membrane is present. (See p. 187.)



FIG. 1.

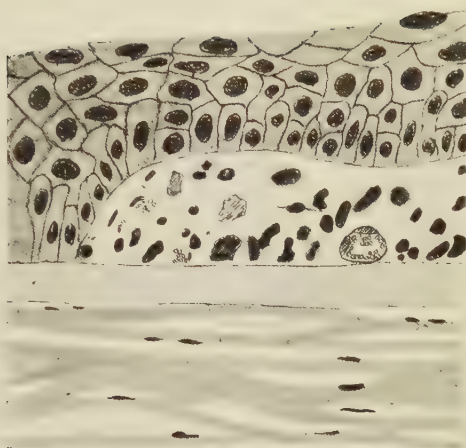


FIG. 2.

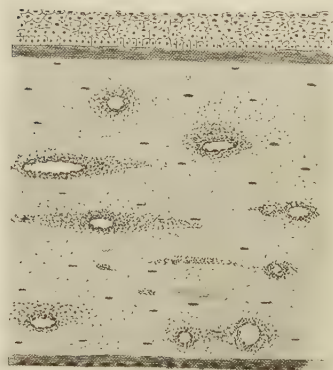


FIG. 4.



FIG. 3.

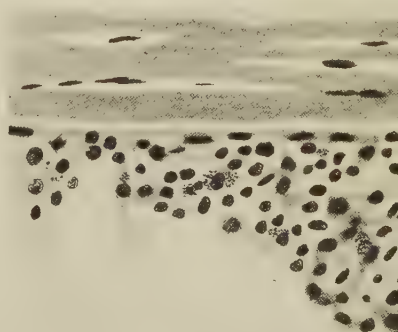


FIG. 5.



PLATE C.

PLATE C.

FIG.

1. *Normal Iris*, showing the pigmented uvea on the posterior surface, and the substantia propria, consisting of connective tissue, blood-vessels, pigment cells, and the constrictor pupillæ muscle (A) near the free extremity. (See p. 172.)
2. *Normal Iris*, the pigment having been removed by bleaching, to disclose the dilator pupillæ muscle (B), not shown in fig. 1, on account of the pigmented uvea. (See p. 172.)
3. *Plastic Iritis*, showing cellular infiltration and thickening of the whole iris, with enlarged blood-vessels, and a deposit of lymph on the anterior surface. (See p. 187.)
4. *Posterior Synechia*, showing almost total attachment of the iris to the anterior capsule (A) of the lens. (See p. 182.)

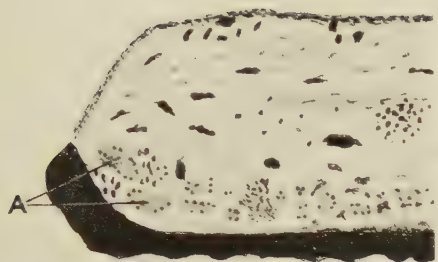


FIG. 1.

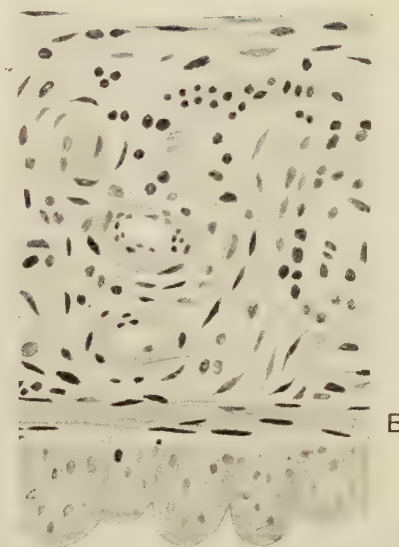


FIG. 2.

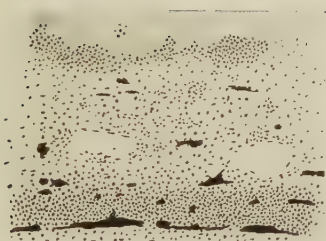


FIG. 3.

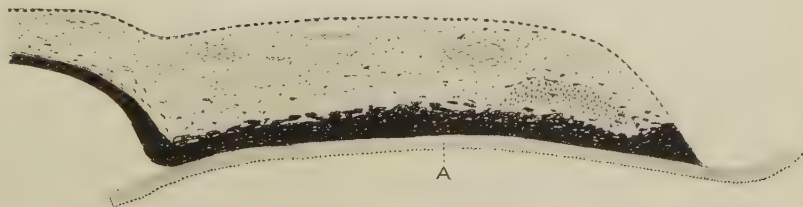


FIG. 4.



PLATE D.

PLATE D.

FIG.

1. *Normal Papilla of the Optic Nerve*, showing the entrance of the optic nerve into the eyeball, the sclerotic opening guarded by the lamina cribrosa, the radiation of the optic-nerve fibres towards the retina, and the physiological cup. (See pp. 242 and 257.)
2. *Glaucomatous Cupping of the Optic Disc*, showing a deep excavation which differs from the physiological cup of fig. 1 in involving the whole diameter of the disc. (See pp. 423 and 424.)

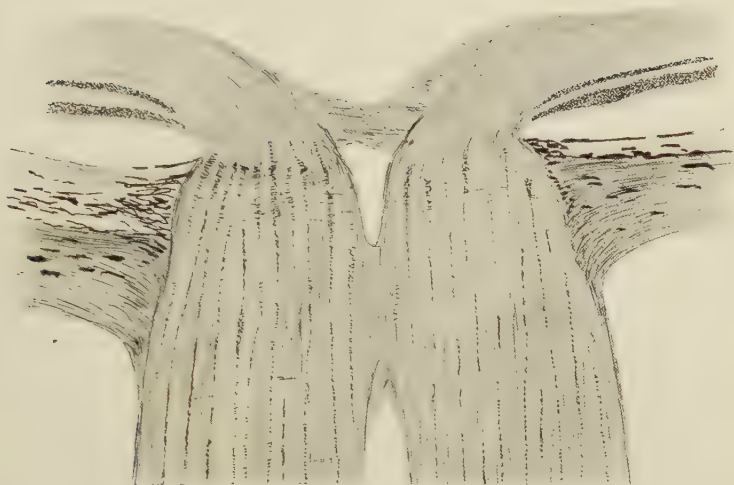


FIG. 1.

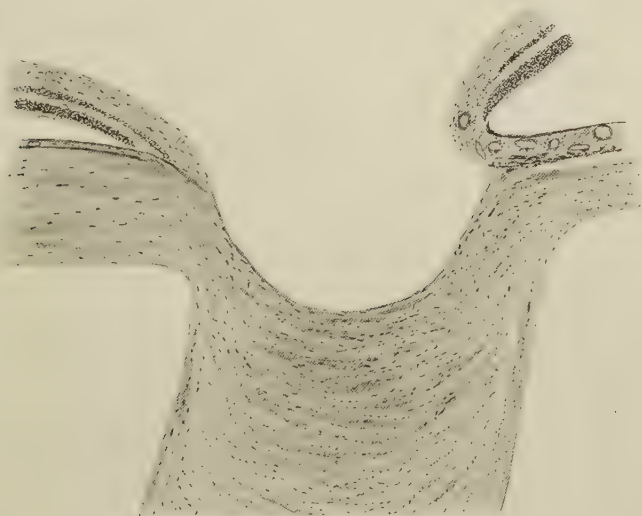


FIG. 2.

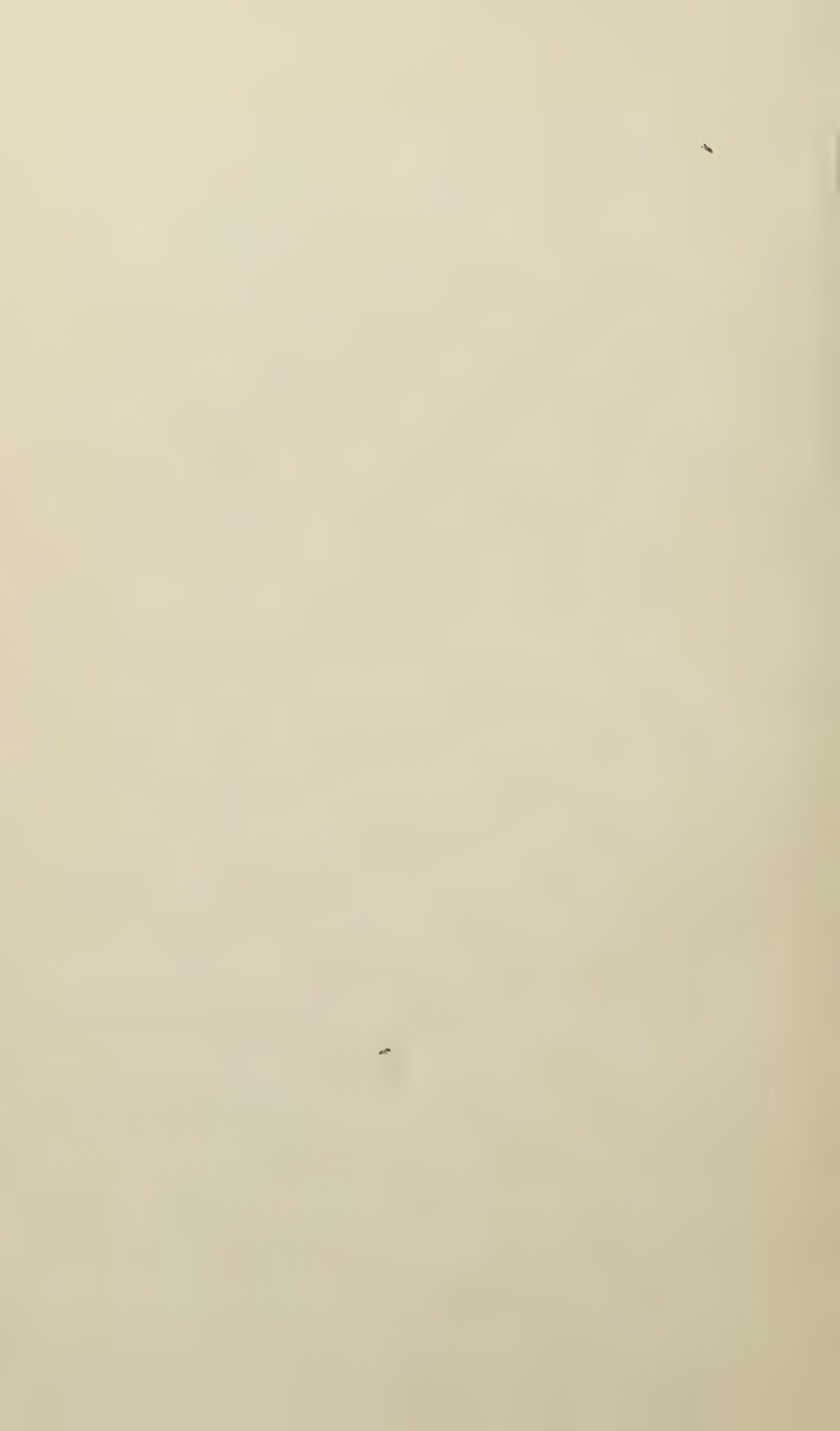


PLATE E.

PLATE E.

FIG.

1. *Normal Choroid*, showing

- a.* Lamina fusca.
- b.* A lymph space.
- c.* Lamina suprachoroidea.
- d.* Lamina vascularis.
- e.* Chorio-capillaris, or membrane of Ruysch.
- f.* Lamina vitrea, or membrane of Bruch.

(See p. 174.)

2. *Exudative Choroiditis*, showing cellular infiltration of the lamina vascularis, and increased thickness of the whole choroid. (See p. 199.)3. *Normal Retina*, showing

- a.* Layer of rods and cones.
- b.* External limiting membrane.
- c.* Outer granular layer.
- d.* Outer molecular layer.
- e.* Inner granular layer.
- f.* Inner molecular layer.
- g.* Ganglionic layer.
- h.* Nerve-fibre layer.

(See p. 244.)

4. *Retinal Hæmorrhages*, showing a large hæmorrhage in the outer molecular layer, and smaller hæmorrhages in all the succeeding layers. (See p. 279.)

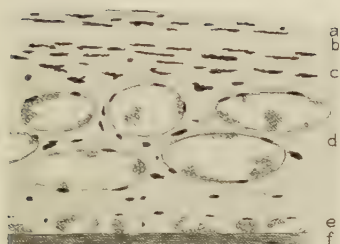


FIG. 1.

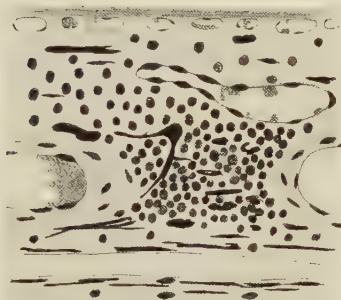


FIG. 2.

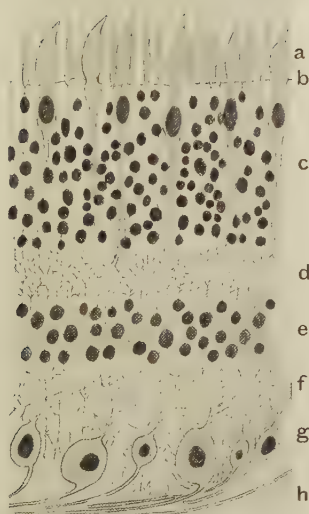


FIG. 3.



FIG. 4.

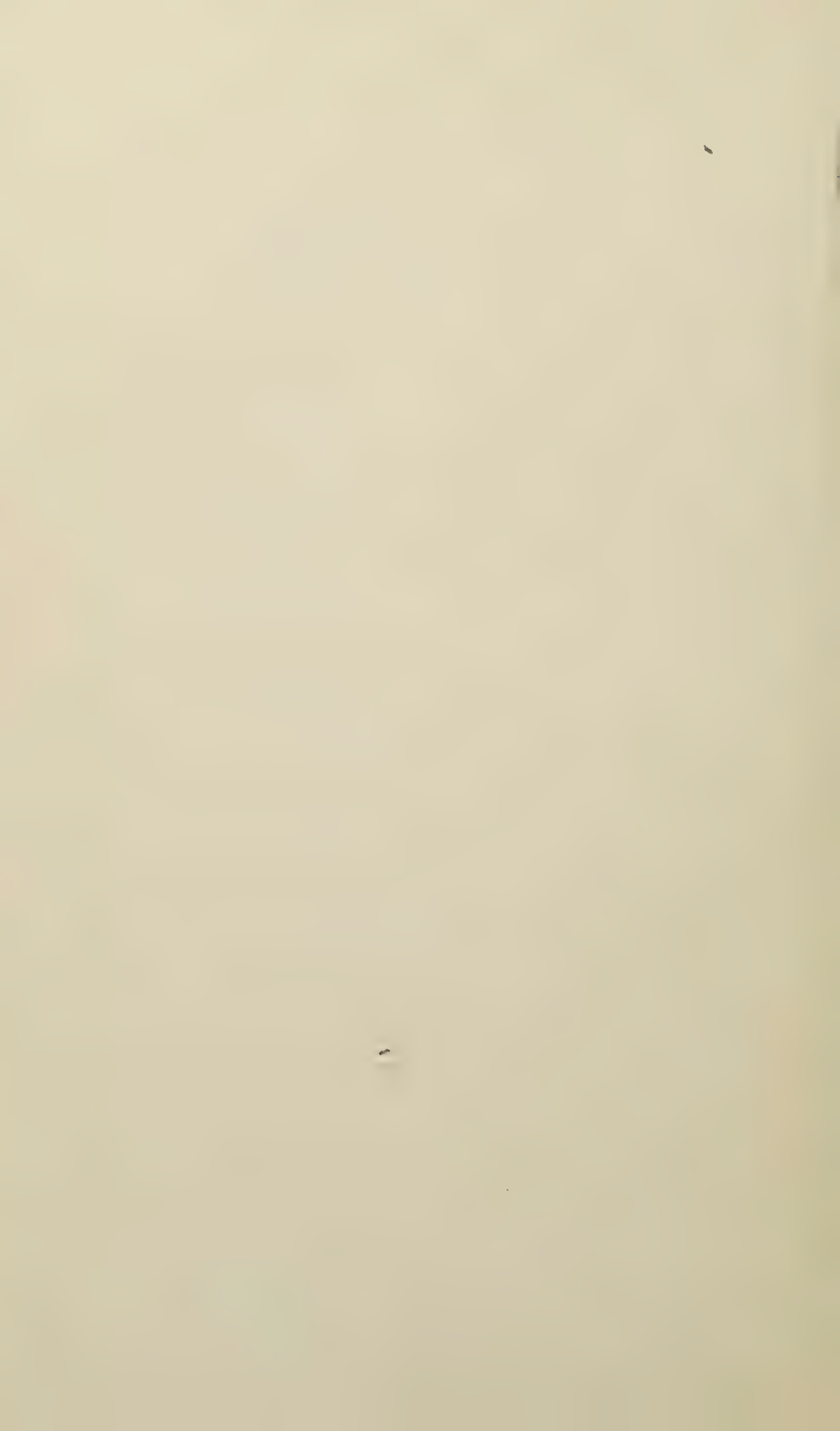


PLATE F.

PLATE F.

FIG.

1. *Normal Lens-fibres.* (See p. 362.)
2. *Incipient Cortical Cataract*, showing opaque cortical fibres. The cubical epithelium lining the anterior portion of the capsule is seen, as well as the marginal transition of this epithelium into lens-fibres. (See p. 365.)
3. *Incipient Nuclear Cataract*, showing the opaque nucleus of the lens. The relative thickness of the anterior and posterior portions of the lens-capsule is indicated, as well as the cubical epithelial lining of the former. (See p. 365.)



FIG. 1.



FIG. 2.



FIG. 3.

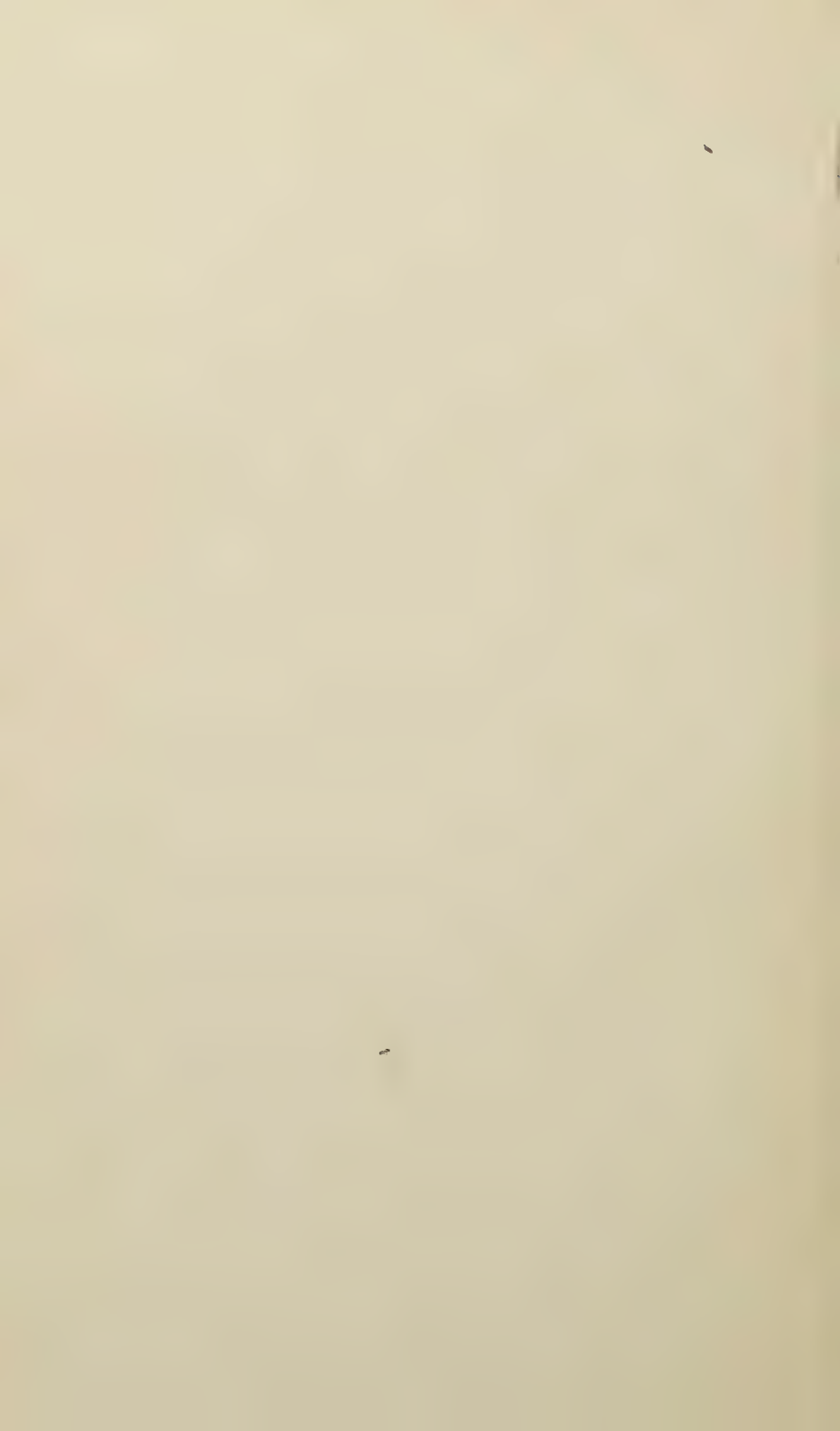


PLATE G.

PLATE G.

FIG.

1. *Lamellar (Zonular) Cataract*, showing a few vacuoles in the centre of the lens, and a considerable number in an intermediate zone; they are absent from the superficial layers of the cortex. (See p. 368.)
2. *Anterior Polar (Pyramidal) Cataract*, showing a structureless opacity with a few cubical epithelial cells scattered here and there within it. Separating this opacity from the transparent lens-fibres is a complete layer of cells, continuous with those lining the anterior capsule elsewhere, and, immediately anterior to these cells, an incomplete homogeneous layer continuous with the anterior capsule itself. (See p. 368.)
3. *Posterior Polar Cataract*, showing irregularly broken-up lens-fibres. (See p. 369.)



FIG. 1.



FIG. 2.

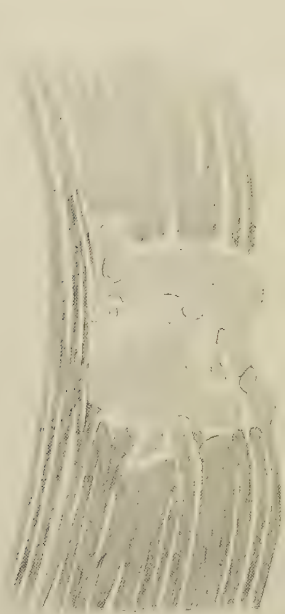


FIG. 3.

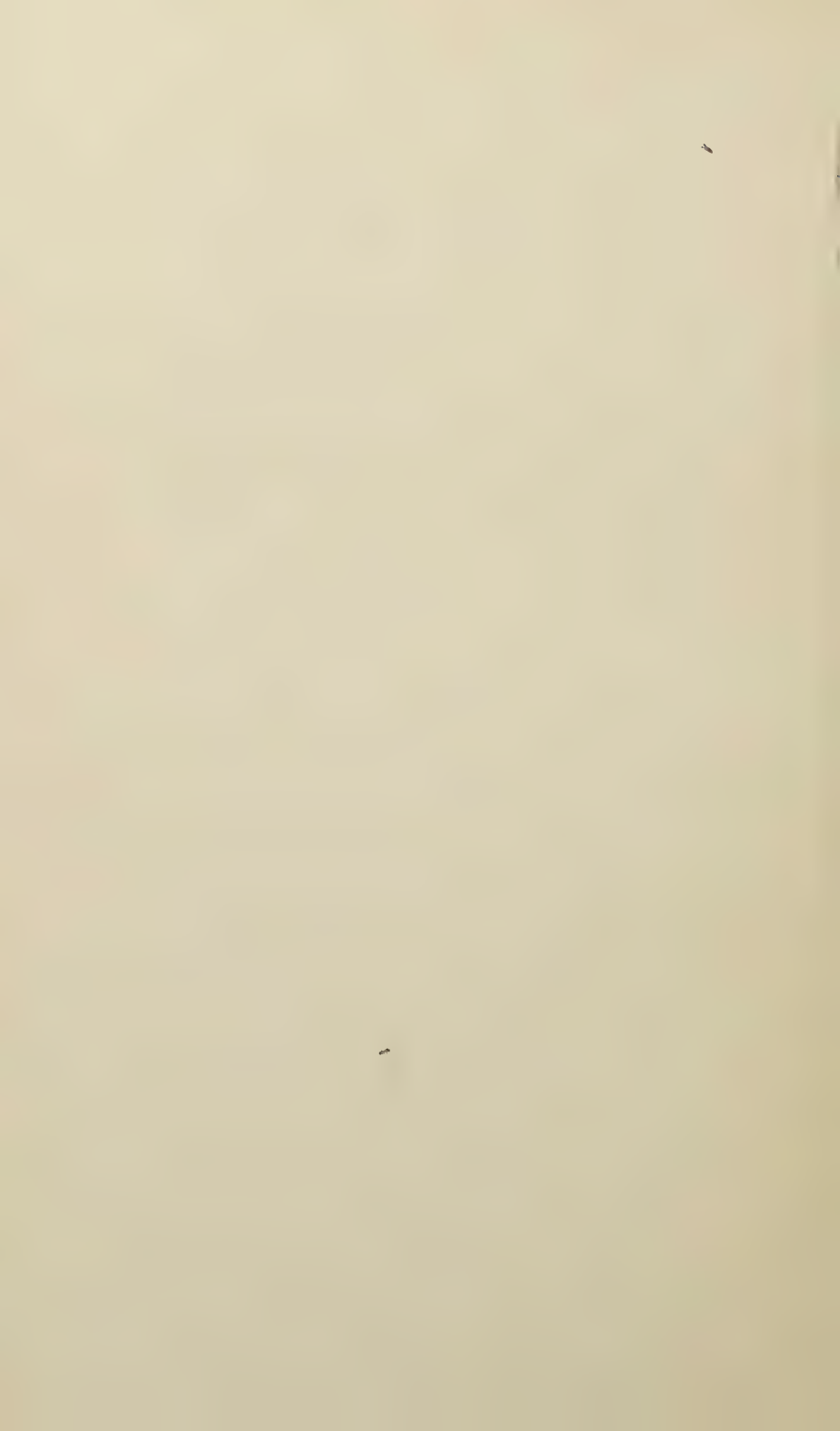


PLATE H.

PLATE H.

FIG.

1. *Normal Ciliary Region*, showing the base of the iris, the roots of two ciliary processes, the ciliary muscle, the sclero-corneal junction with a large Schlemm's canal (*a*). This canal is separated by the ligamentum pectinatum, with the spaces of Fontana, from the widely open iridic angle (*b*). The continuation of the corneal endothelium round the angle and over the anterior surface of the iris is indicated. (See pp. 166, 173, 174.)
2. *Ciliary Region in Early Glaucoma*, showing the same structures as fig. 1. Schlemm's canal (*a*) is constricted and surrounded by cellular infiltration, and the iridic angle (*b*) is partially closed. (See p. 432.)
3. *The same in Late Glaucoma*. Atrophic changes are visible in the iris, the ciliary processes, and round Schlemm's canal (*a*). The iridic angle (*b*) is almost completely closed. (See p. 432.)



FIG. 1.



FIG. 2.

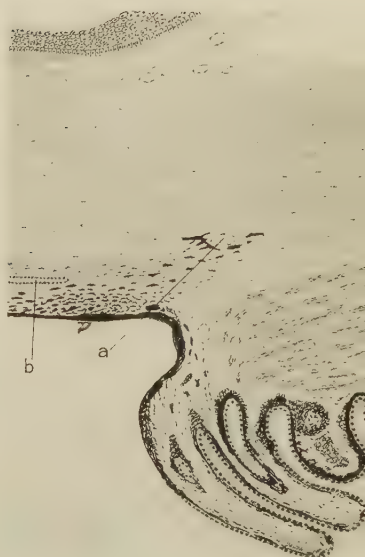
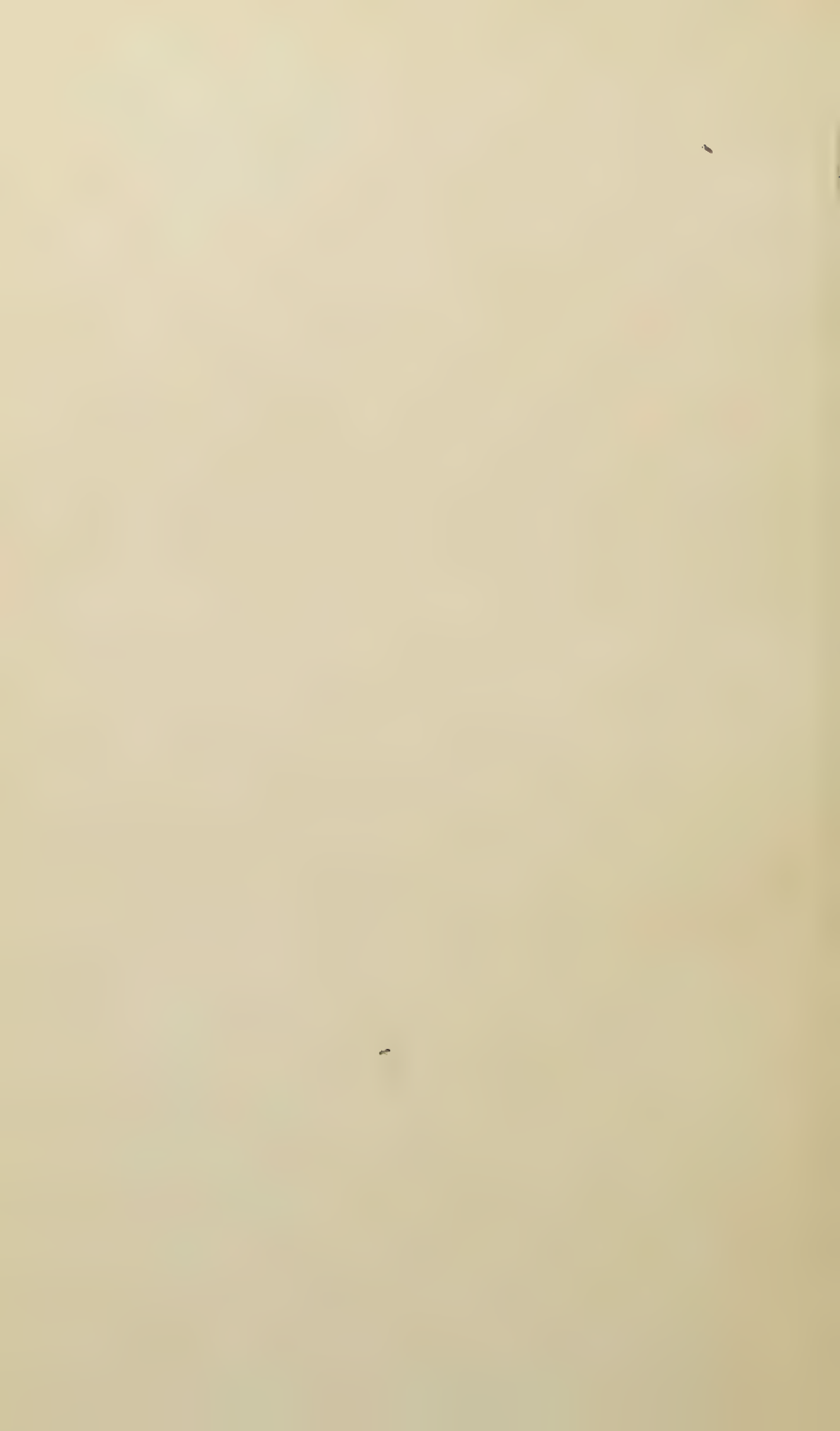


FIG. 3.



GLOSSARY.

<i>Ablepharon</i> . . .	Gr. ἀ privative; βλέφαρον, an eyelid.
<i>Achromatopsia</i> . . .	ἀ privative; χρώμα, colour; ὁράω, I see. Root ὀπ-, fut. ὄψομαι: ὤψ, an eye.
<i>Alexia</i> . . .	ἀ privative; λέξις, a word.
<i>Amacrine</i> . . .	ἀ privative; μακρός, long.
<i>Amaurosis</i> . . .	ἀμαυρόω, I make dark.
<i>Amblyopia</i> . . .	ἀμβλύς, dull; ὁράω, I see.
<i>Ametropia</i> . . .	ἄμετρος, without measure; ὁράω, I see.
<i>Amusia</i> . . .	ἀ privative; μουσα, music.
<i>Aniridia</i> . . .	ἀ privative; ἵρις, a rainbow.
<i>Anisometropia</i> . . .	ἄνισον, unequal; μέτρον, measure; ὁράω, I see.
<i>Ankyloblepharon</i> . . .	ἀγκύλον, crooked; βλέφαρον, an eyelid.
<i>Aphakia</i> . . .	ἀ privative; φακός, a lentil.
<i>Argyriasis</i> . . .	ἄργυρος, silver.
<i>Asthenopia</i> . . .	ἀ privative; σθένος, power; ὁράω, I see.
<i>Astigmatism</i> . . .	ἀ privative; στίγμα, a point.
<i>Blennorrhœa</i> . . .	βλέννα, mucus; ῥέω, I flow.
<i>Blepharitis</i> . . .	βλέφαρον, an eyelid.
<i>Blepharophimosis</i> . . .	βλέφαρον, an eyelid; φίμωσις, contraction.
<i>Buphthalmos</i> . . .	β. ūs, a bull; ὀφθαλμός, an eye.
<i>Canthoplasty</i> . . .	κανθός, angle of the eye; πλάσσω, I form.
<i>Caruncle</i> . . .	L. dim. of caro, flesh.
<i>Cataract</i> . . .	Gr. καταρραγῆναι, to burst down; from its supposed resemblance to the foam of a large cascade.
<i>Chalazion</i> . . .	χάλαζα, a hailstone.
<i>Chemosis</i> . . .	χήμη, a cockle-shell.
<i>Choroid</i> . . .	τὸ χωρίον, the chorion; εἶδος, likeness.
<i>Coloboma</i> . . .	κολόβωμα, a deficiency.
<i>Corectopia</i> . . .	κόρη, a pupil; ἔκτοπος, out of place.
<i>Cornea</i> . . .	L. corneus, horny.
<i>Crypto-glioma</i> . . .	Gr. κρυπτός, hidden; γλία, glue.
<i>Cryptophthalmos</i> . . .	κρυπτός, hidden; ὀφθαλμός, an eye.
<i>Cyclitis</i> . . .	κύκλος, a circle.
<i>Cycloplegia</i> . . .	κύκλος, a circular body; πλῆσσω, I paralyse.
<i>Dacryo-adenitis</i> . . .	δάκρυ, a tear; ἀδὴν, a gland.
<i>Dacryops</i> . . .	δάκρυ, a tear; ὁράω, I see.

<i>Dioptre</i> . . .	Gr. διά, through; ὁράω, I see.
<i>Diplopia</i> . . .	δίς, double; ὁράω, I see.
<i>Distichiasis</i> . . .	δίς, double; στίχος, a row.
<i>Dyslexia</i> . . .	δυσ, difficult; λέξις, a word.
<i>Ectropion</i> . . .	ἐκτρέπω, I evert.
<i>Emmetropia</i> . . .	ἔμμετρος, in measure; ὁράω, I see.
<i>Encanthis</i> . . .	ἐν, in; κανθός, angle of the eye.
<i>Enophthalmos</i> . . .	ἐν, in; ὀφθαλμός, an eye.
<i>Entropion</i> . . .	ἐντρέπω, I turn in.
<i>Epicanthis</i> . . .	ἐπί, on; κανθός, angle of the eye.
<i>Epiphora</i> . . .	ἐπιφέρεισθαι, to rush upon.
<i>Erythropsia</i> . . .	ἐρυθρός, red; ὁράω, I see.
<i>Esophoria</i> . . .	ἔσω, inwards; φέρω, I bear.
<i>Exophoria</i> . . .	ἔξω, outwards; φέρω, I bear.
<i>Exophthalmos</i> . . .	ἐκ, out; ὀφθαλμός, an eye.
<i>Glaucoma</i> . . .	γλαυκός, green.
<i>Glioma</i> . . .	γλία, glue.
<i>Hemeralopia</i> . . .	ἡμέρα, day; ἀλαός, blind; ὁράω, I see.
<i>Hemianopsia</i> . . .	ἡμισυς, half; ἀ, privative; ὁράω, I see.
<i>Heterochromia</i> . . .	ἕτερον, another; χρώμα, colour.
<i>Heterophoria</i> . . .	ἕτερον, another; φέρω, I bear.
<i>Heterophthalmos</i> . . .	ἕτερον, another; ὀφθαλμός, an eye.
<i>Hippus</i> . . .	ἵππος, a horse.
<i>Hordeolum</i> . . .	L. dim. of <i>hordeum</i> , barley.
<i>Hyalitis</i> . . .	Gr. ὕαλος, glass.
<i>Hydrophthalmos</i> . . .	ὑδωρ, water; ὀφθαλμός, an eye.
<i>Hypermetropia</i> . . .	ὑπέρ, in excess; μέτρον, standard; ὁράω, I see.
<i>Hyperphoria</i> . . .	ὑπέρ, in excess; φέρω, I bear.
<i>Hyphæma</i> . . .	ὑπό, beneath; αἷμα, blood.
<i>Hypopyon</i> . . .	ὑπό, beneath; πύον, pus.
<i>Irideremia</i> . . .	ἶρις, a rainbow; ἐρημία, absence of.
<i>Iridodesis</i> . . .	ἶρις, a rainbow; δέω, I tie.
<i>Irido-dialysis</i> . . .	ἶρις, a rainbow; διαλύω, I part asunder.
<i>Iridodonesis</i> . . .	ἶρις, a rainbow; δονέω, I shake.
<i>Iridoplegia</i> . . .	ἶρις, a rainbow; πλίσσω, I paralyse.
<i>Iris</i> . . .	ἶρις, a rainbow.
<i>Keratitis</i> . . .	κέρας, a horn.
<i>Keratocoele</i> . . .	κέρας, a horn; κήλη, rupture.
<i>Keratomalacia</i> . . .	κέρας, a horn; μαλακόν, soft.
<i>Lagophthalmos</i> . . .	λαγός, a hare; ὀφθαλμός, an eye.
<i>Lenticonus</i> . . .	L. <i>lens</i> , lens; <i>conus</i> , cone.
<i>Leucoma</i> . . .	Gr. λευκός, white.
<i>Macula</i> . . .	L. <i>macula</i> , a spot.
<i>Megalopsia</i> . . .	Gr. μέγας, great; ὁράω, I see.
<i>Metamorphopsia</i> . . .	μεταμόρφωσις, a transformation; ὁράω, I see.
<i>Microphthalmos</i> . . .	μικρός, small; ὀφθαλμός, an eye.
<i>Micropsia</i> . . .	μικρός, small; ὁράω, I see.

<i>Miosis</i>	. . .	Gr. μείωσις, contraction.
<i>Myopia</i>	. . .	μύω, I close; ὀράω, I see.
<i>Nebula</i>	. . .	L. <i>nebula</i> , mist.
<i>Nyctalopia</i>	. . .	Gr. νύξ, night; ἀλαός, blind; ὀράω, I see.
<i>Nystagmus</i>	. . .	νυστάζω, I nod sleepily.
<i>Onyx</i>	. . .	ὄνυξ, a nail.
<i>Ophthalmia</i>	. . .	ὀφθαλμός, an eye.
<i>Orthophoria</i>	. . .	ὀρθός, straight; φέρω, I bear.
<i>Palpebra</i>	. . .	L. <i>palpo</i> , I strike.
<i>Pannus</i>	. . .	<i>pannus</i> , a cloth.
<i>Panophthalmitis</i>	. . .	Gr. πᾶς, all; ὀφθαλμός, an eye.
<i>Pemphigus</i>	. . .	πέμφιξ, a blister.
<i>Phlyctenule</i>	. . .	dim. of φλύκταινα, a vesicle.
<i>Photophobia</i>	. . .	φῶς, light; φόβος, fear.
<i>Photopsia</i>	. . .	φῶς, light; ὀράω, I see.
<i>Phtheiriasis</i>	. . .	φθείρ, a louse.
<i>Pinguecula</i>	. . .	L. dim. of <i>pinguis</i> , fat.
<i>Polycoria</i>	. . .	Gr. πολὺς, many; κόρη, a pupil.
<i>Presbyopia</i>	. . .	πρέσβυς, old; ὀράω, I see.
<i>Proptosis</i>	. . .	προπίπτω, I protrude.
<i>Pterygium</i>	. . .	πτέρυξ, a wing.
<i>Ptosis</i>	. . .	πίπτω, I fall.
<i>Retina</i>	. . .	L. <i>rete</i> , a net.
<i>Rhodopsia</i>	. . .	Gr. ῥόδον, rose; ὄψις, vision.
<i>Sclerotic</i>	. . .	σκληρός, hard.
<i>Scotoma</i>	. . .	σκότος, darkness.
<i>Staphyloma</i>	. . .	σταφυλή, a bunch of grapes.
<i>Strabismus</i>	. . .	στρέφω, I turn.
<i>Stye</i>	. . .	Anglo-S. <i>stigan</i> , to spring up.
<i>Sycosis</i>	. . .	Gr. σῦκον, a fig.
<i>Symblepharon</i>	. . .	σύν, together; βλέφαρον, an eyelid.
<i>Synchysis</i>	. . .	σύν, together; χέω, I pour.
<i>Syndectomy</i>	. . .	σύν, together; τέμνω, I cut.
<i>Synechia</i>	. . .	σύν, together; ἔχω, I hold.
<i>Tarsorrhaphy</i>	. . .	ταρσός, tarsus; ραφή, a suture.
<i>Trachoma</i>	. . .	τραχύς, rough.
<i>Trichiasis</i>	. . .	θρίξ, a hair.
<i>Uvea</i>	. . .	L. <i>uva</i> , an unripe grape.
<i>Xanthelasma</i>	. . .	Gr. ξανθός, yellow; ἔλασμα, a layer.
<i>Xerophthalmos</i>	. . .	ξηρός, dry; ὀφθαλμός, an eye.
<i>Xerosis</i>	. . .	ξηρός, dry.



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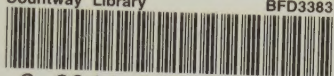


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